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ARTIFICIAL ELONGATION OF TEETH

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AS FAR as I know no human or other specimen was ever published concerning the histologic changes occurring during artificial elongation of teeth except the specimen of a monkey tooth shown in my first publication on tissue changes during orthodontic movement.[3a] This specimen showed that the stimulus transmitted through the alveolar ridge fibers to the alveolar crest resulted in its growth and development. The bony spicules followed this traction, became elongated and formed new bone at their ends facing the alveolar crest, while they became resorbed at the other ends. This same stimulus was also transmitted to the bone forming the alveolus as well as to that at the fundus of alveolus; thus the whole bone involved was induced to follow the occlusal movement of the tooth; the bony spicules became extended in the direction of the force being surrounded at the points of the attachment of the periodontal fibers by osteoid bone and numerous osteoblasts.[3, 3a] This formation of new bone is in accordance with nature's endeavor to maintain the firmness of the tooth that otherwise would become always more loose in the same degree as the smaller diameter of the root comes in a wider diameter of the socket. By the apposition of new bone the widened periodental membrane becomes reduced again to nearly its normal width; in several instances it even may become narrower than normal by overproduction of new bone overshooting the mark.

We are familiar with this phenomenon of overshooting the mark almost regularly found on the pressure side; here it serves to overcome at a quicker rate the undue pressure by widening the periodontal space more than necessary for the immediate need of the movement. This explains the fact that in nonbiologic tooth movements if the forces are not too strong to crush the tissues the width of the periodontal membrane generally is found wider on the pressure side.

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It was shown by the human specimens[3b] that the transmission of a light stimulus of traction, where no fibers were torn, was responsible for the fact that the newly built osteoid bone was formed in all cases in a uniformly thick layer as shown in Fig. 1, whereas in application of stronger forces the picture changes entirely as in Fig. 2. In such cases where several fibers are torn loosening, thereby, their points of attachment either on the bone or on the cementum surface, the transmission of the stimulus to the bone was partly eliminated, and the bone responded with osteoid formation only on those places where the fibers have remained uninjured. Therefore we find an uneven bone surface, ridges of osteoid bone alternating with grooves corresponding to regions of intact and torn fibers respectively.

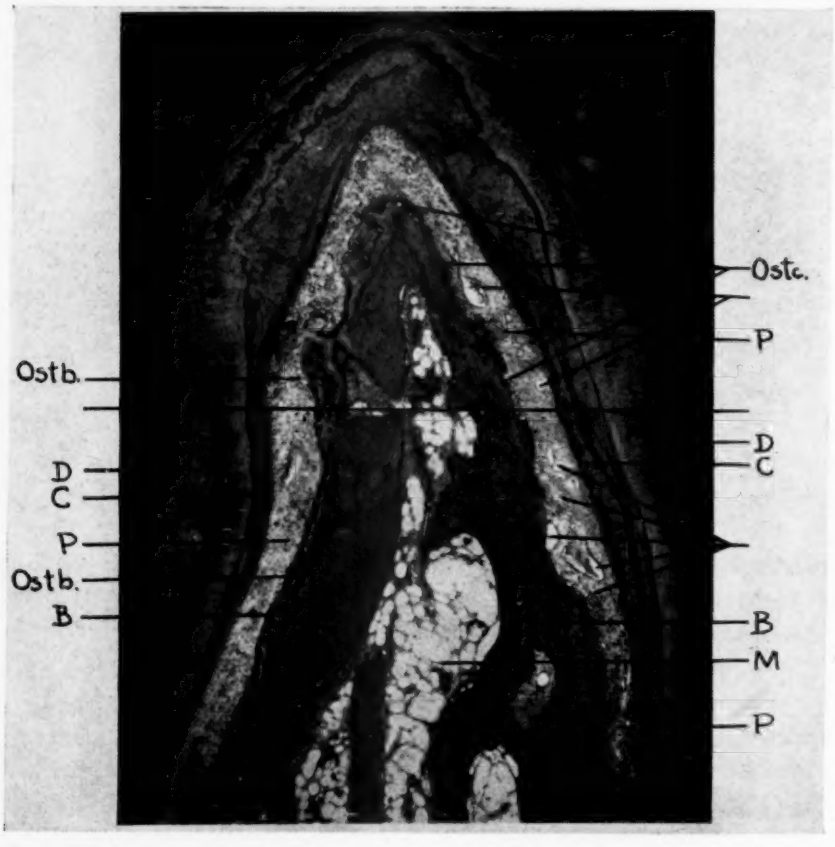

Fig. 1.—Interradical septum; on the left traction side. On the right pressure side; *Ostb.*, osteoid; *Ostc.*, osteoclasts; *P*, periodontal membrane; *D*, dentin; *C*, cementum; *B*, bone; *M*, bone marrow. (From the *Angle Orthodontist.*)

According to my conception, the even apposition of bone has to be considered as *more* biologic for it is found oftentimes in teeth that after artificial movements relapsed toward their original position without appliance interference, while the formation of spicules, as mentioned already, is found only in the use of relatively strong forces.

The apposition of osteoid bone on the traction side in an even layer is considered by me only as *more* biologic than the formation of spicules, for as real biologic bone reaction one can consider only the formation of bundle bone. And, as such bone was never shown in artificial tooth movements, we were

justified also by this negative finding, besides others, to state that there does not exist a biologic orthodontic tooth movement.

It is also the author's belief that the formation of osteoid bone in an even layer may be favorable in reducing the time of retention, for here there are no "grooves" that have to be filled up with bone to form again a normal smooth bone surface of the socket. The reaction of the bone and the other involved tissues to strong elongating forces is unknown.

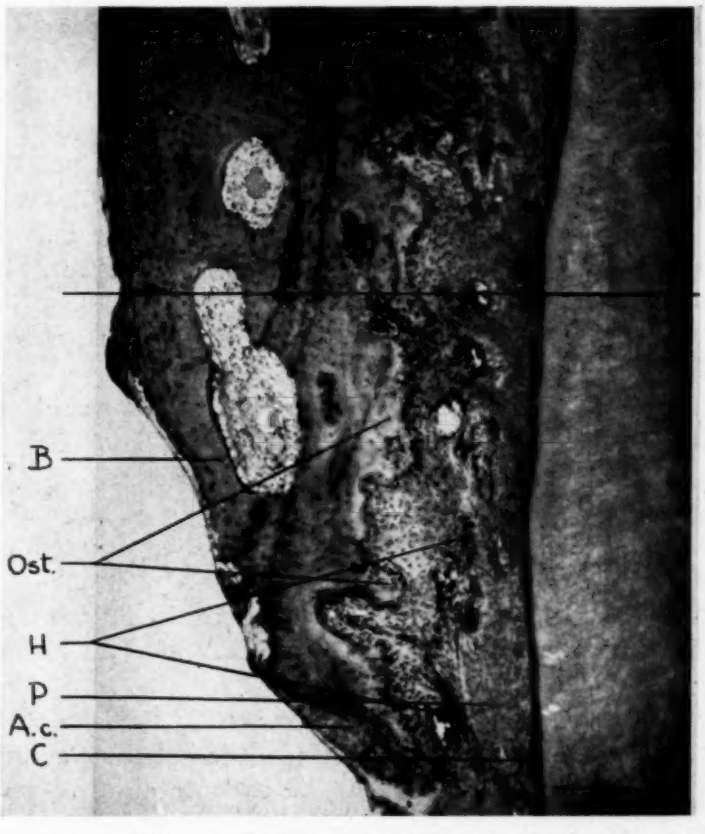

Fig. 2.—*B*, bone; *Ost*, osteoid bone; *H*, hemorrhages; *P*, periodontal membrane; *C*, cementum; *A.c.*, alveolar crest; Arrow, direction of movement. (From the *Angle Orthodontist.*)

We don't know whether the bone at the alveolar crest fails to follow the traction of the fibers on account of their breakage, exposing the cementum, nor do we know anything about the bone reaction on the sides of the root and at the fundus. We may only assume a pathologic condition of the soft tissues (breakage of fibers, hemorrhages) and a disproportionate width of the periodontal space, for a smaller diameter of the root comes into a wider diameter of the socket. This condition becomes still more aggravated by the anatomic peculiarity in the course of the periodontal fibers. Normally the fibers have an oblique course, their points of attachment on the bone lying in a higher level toward the alveolar crest and descending toward the root to their attachment points in the cementum. By elongation these fibers become therefore relaxed, and no stimulus at first can be transmitted to the bone. Such a stimulus is created probably later when the fibers with the progress of the movement become stretched again like in the monkey tooth[3a] where the movement lasted a

relatively long time, namely forty days; according to Marshall[5] this interval is supposed to correspond to two hundred days in man.

After the completion of such a movement of course a whole rearrangement of the tissues and especially of the fibers has to take place for their course is now opposite to normal; the points of the fiber attachment on the root are now nearer the alveolar crest and give no support whatever to the tooth. We don't know anything at present about the morphologic changes of this rearrangement nor about the time necessary for it, although this is of major importance in retention.

That there must exist in the use of strong forces a wide periodontal membrane, induced by the discrepancy between the diameter of the root and the corresponding socket, can as yet only be guessed at by the clinical evidence of the great looseness of these teeth, for we do not have any histologic evidence. Such a looseness never is encountered in teeth where only other than elongating movements were performed. The relaxation of the fibers of the periodontal membrane happens, of course, also in slow movement, and we will see later the reaction of the bone under this condition.

To show the reaction of the tissues involved in this movement in the use of light forces is the purpose of this paper. It deals with a mandibular premolar of a 12-year-old girl which was elongated about 1½ mm. by a tiny spring soldered to a lingual arch. The tooth was banded and provided with a lingual spur under which the spring was fastened with a ligature. The spring was 1½ cm. long and 0.25 mm. thick and was active over a distance of one-half mm. corresponding to the thickness of the spur on the band. The tooth was in occlusion and the space for the intended movement was procured by grinding the occlusal surface every two weeks. This was repeated five times and two weeks after the last adjustment the tooth was extracted. The whole movement lasted twelve weeks.

The nonbiologic changes that we encounter on the root surface were brought about by tooth movements that were not intended; but these movements, caused by the occlusal forces, cannot be eliminated and are always present except in teeth without antagonists.

The normal occlusal forces have to act pathologically under the existing conditions for the elongated tooth is loose; it lies in a socket far too wide until nature provides appropriate countermeasures reducing the pathologic width of the periodontal membrane to normal.

In Fig. 3 we see the low magnification of the tooth with some of the buccal alveolar bone attached, *A;* the deep loss of substance, *B,* is artificial and was caused during the removal of the tooth; in *C* we see an indication of and in *D, E,* and *F* well-developed cement resorptions reaching into the dentine. The alveolar crest is covered with a cap of new formed osteoid bone (Fig. 4*A*) surrounded by numerous osteoblasts (Fig. 4, *Ostb.*) a high magnification of the alveolar crest of Fig. 3. The bone followed the occlusal movement of the tooth, increasing thereby the height of the alveolar crest. The reaction of the bone at the apex cannot be ascertained for it is not present in the specimen. The surface of the bone facing the tooth shows the signs of apposition, a quite thin strip of osteoid bone covered with osteoblasts (Fig. 4, *Ost.*). This bone formation did not happen under the stimulating influence of the periodontal fibers,

for they were relaxed; we do not find elongated bony spicules such as are found on the traction side of a tooth under the influence of heavier forces causing, as explained above, the formation of an irregular bone surface.

The "passive" apposition of bone that happens without transmission of a stimulus through stretched fibers follows only the creation of a wider than normal periodontal space. The fibers of the periodontal membrane do not even show any sign of functional arrangement; they have a weavy appearance but mostly their course cannot be determined. This relaxed condition of the fibers may account for the slow and even apposition of the bone.

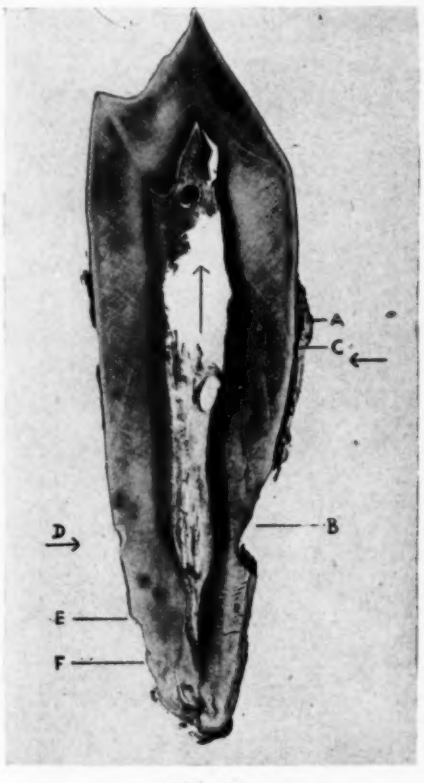

Fig. 3.

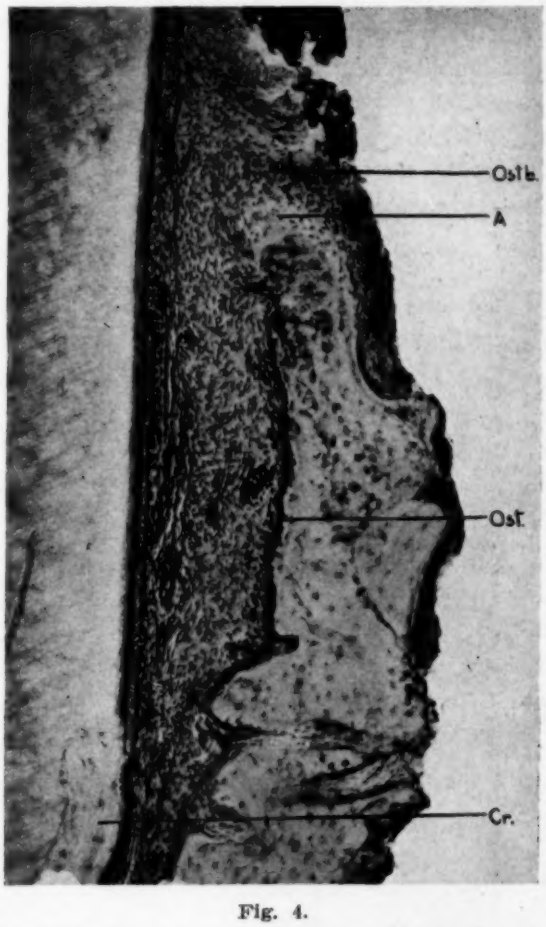

Fig. 4.

Fig. 3.—Low magnification; *A*, buccal alveolar bone; *B*, artificial damage; *C*, *D*, *E*, *F*, cement resorptions; Arrow, direction of movement.

Fig. 4.—Alveolar crest, *A* (high magnification), covered with a cap of osteoid bone and surrounded by osteoblasts, *Ostb.*; *Ost.*, seam of osteoid bone; *Cr.*, cement resorption.

The cement resorptions buccally near the alveolar crest and lingually near the apex may be attributed to the relaxation of the fibers and the great width of the periodontal membrane. Thereby the resistance power of the tooth is diminished to a high degree and with each occlusal contact the tooth is pushed and tipped against the bone, finding here a fulcrum whereby the apex is forced to move in the opposite direction. The compression of the periodontal membrane lasting only a short time and happening intermittently does not cause a crushing of the tissues or necrosis of the cells; they have opportunity to re-

cover very quickly and, therefore, their vitality does not become lost or even impaired; they can be transformed into osteoblasts or osteoclasts and cementoblasts or cementoclasts as the case may be.

Osteoid bone is formed during the rest periods in the use of intermittent forces and is supposedly more resistant to resorption. But for the creation of the resorptions shown here as in the other human specimens[3b] the resistance of the normal bone is sufficient. The shown specimen is another proof of my contention that the human cementum is a quite vulnerable structure and it is the more a proof as these resorptions were caused by movements that never were intended; they represent unexpected occurrences brought about by secondary movements accompanying the main intended movement. This vulnerability of the human cementum is also corroborated by the statement of Euler-Mayer[1] (page 264); "the abundant occurrence of cementum resorptions is verified by the fact that we seldom encounter a functioning tooth that does not show in its cementum the vestiges of a once existing resorption."

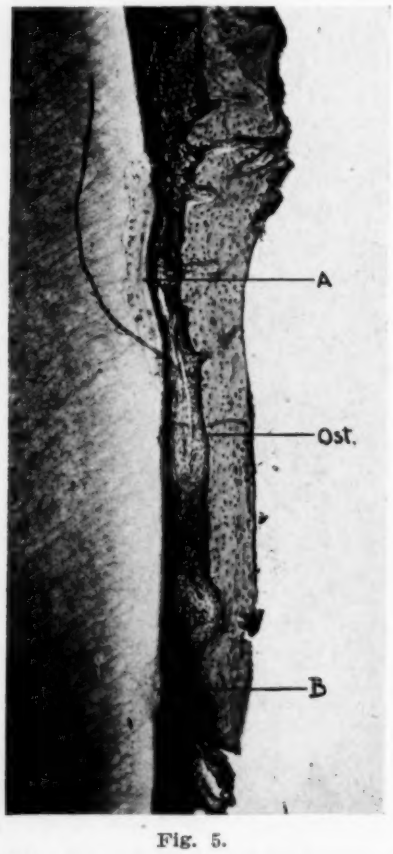

Fig. 5.

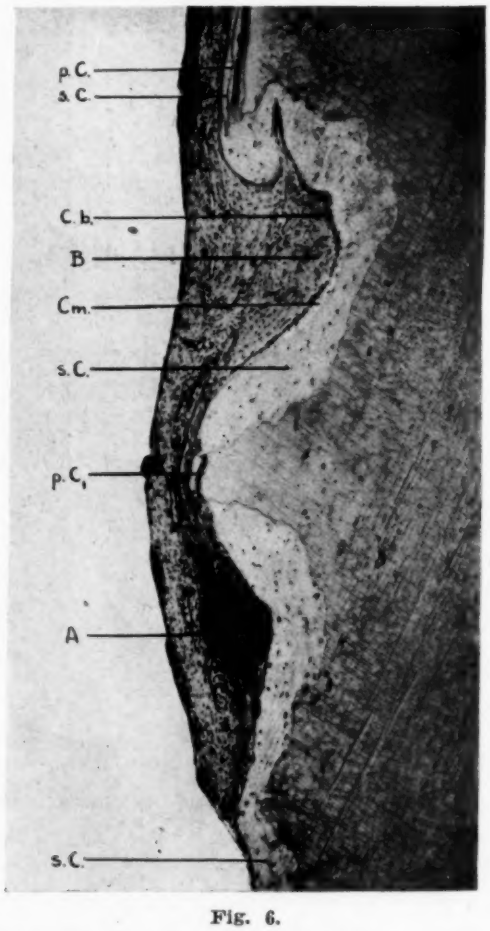

Fig. 6.

Fig. 5.—Cement resorptions, corresponding to "*C*" in Fig. 3; *Ost.*, osteoid bone; covered by osteoblasts; *A*, *B*, cement resorptions, smoothed by secondary cementum deposition.

Fig. 6.—Cement resorptions in the apex region corresponding to "*D*" in Fig. 3; secondary cementum (*s.C.*) overflowing the borders covering for some distance the primary cementum (*p.C.*); cementoblast, *Cb.*; cementoid, *Cm.*

At the time of the extraction all resorptions were in a more or less advanced state of repair. Secondary cementum has filled up entirely the resorp-

tions near the alveolar crest shown in Fig. 5*A* and *B;* the upper part of the resorption, *A*, is also seen in Fig. 4, *Cr*. This deposition of cementum is not so far progressed in the resorptions near the apex in Fig. 6. This picture, a high magnification of *D* in Fig. 3, is taken from a neighboring slide. It shows two deep resorptions; one is nearly repaired and smoothed by secondary cement deposition (Fig. 6*A*) while the second and larger resorption (Fig. 6*B*) is not yet completely repaired, but the cementum bears the signs of great vitality for its surface is lined with a stripe of cementoid (*Cm.*) covered with many cementoblasts (Fig. 6, *Cb.*). This secondary cementum (*s.C.*) overflows the margins of the resorption covering, for some distance, the primary cementum (Fig. 6, *p.C.*). Between the two resorptions remained an isle of primary cementum (*p.*C_1.).

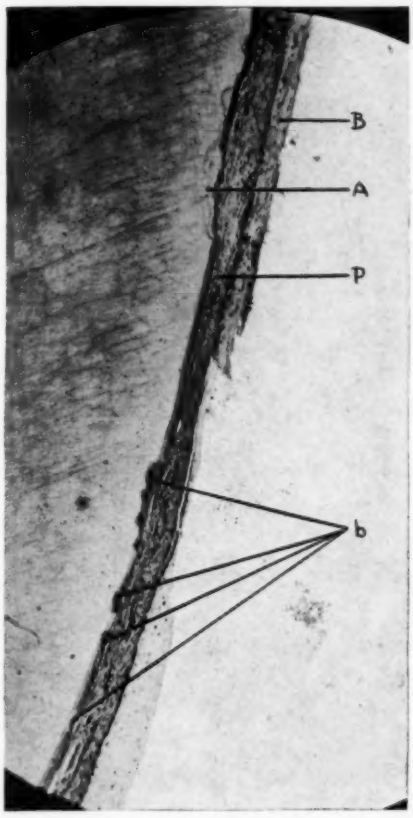

Fig. 7.—*A, b,* cement resorptions in different state of repair; *A,* repaired; *B,* bone; *P,* periodontal membrane.

In another section of the same specimen from the same region of the buccal alveolar wall as the resorptions shown in Fig. 5, we find several smaller resorptions in various stages of repair; while the greatest and uppermost nearest the alveolar ridge is nearly repaired (Fig. 7*A*), the smaller ones (Fig. 7*B*) are just about to be repaired.

It is a very well-established fact that cement resorptions generally have a great healing tendency and that the repair starts as soon as the cause is eliminated either by the discontinuance of the force or by the disappearance of the bone against which the root was leaning. On the other hand it cannot be denied

that in some instances such a healing tendency does not exist, but on the contrary a very powerful penetrating tendency may exist, by which the pulp may become exposed and a communication established between the pulp and the periodontal membrane.[3] Such an occurrence may not happen very often in the use of strong continuous forces, but the possibility of such a fact ought not to be excluded.

It may be that constitution or a faulty metabolism is responsible; but this does not tell us much as long as we don't know how to successfully check this disturbance. The possibility of a lack of the healing tendency is also admitted by Gottlieb-Orban[2] and Orban,[4] respectively, in their works.

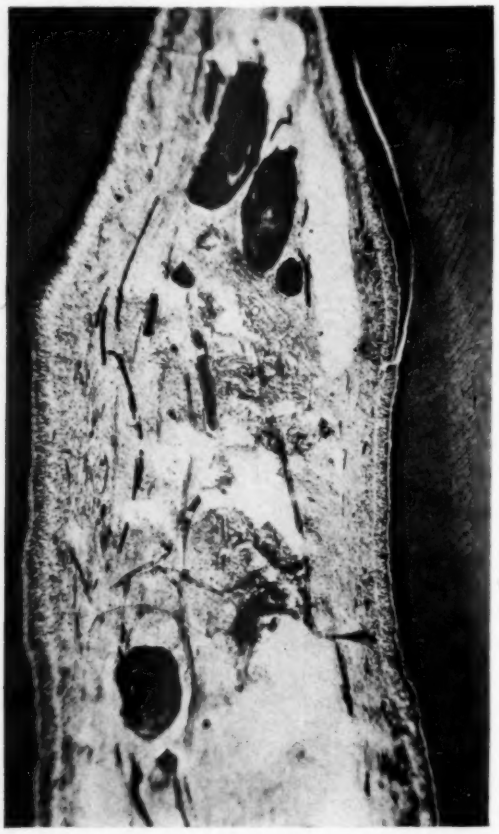

Fig. 8.—Pulp chamber with six pulp stones of different size.

The former say "that each resorption on the tooth must be regarded as a danger to the tooth" and "tooth resorptions are on the whole incalculable"; Orban says, "Sometimes resorptions are not repaired but may continue until the tooth becomes completely detached and lost."

The pulp condition was also not found normal as in all orthodontically moved teeth examined up to now. This fact of damage of the pulp is acknowledged by nearly all authors who had human material at their disposal. But this specimen shows a disturbance limited almost entirely to a deterioration of the calcium metabolism, a condition not heretofore observed so clearly.

It is not uncommon to encounter denticles in children's teeth after orthodontic interference; in the human specimens examined I could find them in

three cases, that is in 21.4 per cent. Whether the orthodontic movement may be considered as the trauma to bring about this precocious precipitation of calcium salts similar to traumatic interferences in the adult, which are considered by some authors as the cause of the formation of denticles, is still an unsolved question.

The more or less pronounced damages of the pulp tissue encountered in previously examined cases[3b] were in this case only slight. In the pulp chamber (Fig. 8) taken from a neighboring section, the vessels are not very much increased in number and diameter; only several of them are thrombosed. Generally the pulp tissue has preserved its embryonic character. The most significant feature very seldom found in other specimens of treated teeth is the undisturbed row of odontoblasts; the formation of very small vacuoles between

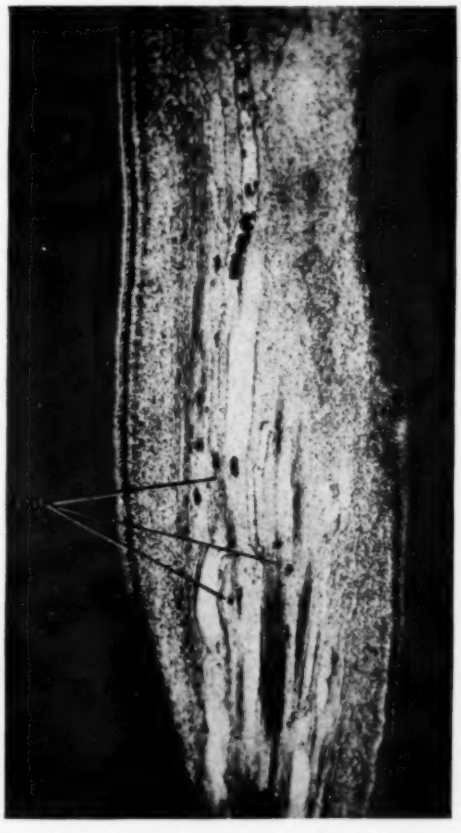

Fig. 9.—Root pulp with several calcospherites (*Ca.*).

them is encountered only now and then. The dentine is formed in normal width, the sign of the normal activity of the odontoblasts; the only striking pathologic appearance, as already mentioned, is the presence of several larger and smaller denticles. In the root pulp they are in the initial stage of formation though quite numerous (Fig. 9, *ca*). It seems as if the pulp has suffered no greater damage than that it has lost its normal calcium metabolism, has lost its faculty to "prevent the precipitating of the dissolved chalk" (Euler-Mayer).[1] One cannot tell whether this faculty can be regained after the causative factor has subsided or whether this process continues and ends in the calcification of the pulp. The ultimate destiny of every pulp once damaged remains an open ques-

tion whether brought about by orthodontic intervention or otherwise. While some authors believe that the damages can be overcome if the conditions of metabolism improve, there are others who maintain that any damage of the pulp brought about by disturbances of circulation and advanced to a certain degree leads irrevocably sooner or later to the death of the pulp.

Of course one should not draw conclusions from one specimen; further investigation is necessary. This should comprise not only material where light forces, as in our case, were used but also especially where stronger and really strong forces were applied, e.g., the force of elastics or springs to bring into alignment high standing canines or to elevate premolars in straightening the curve of Spee. As far as my own experience is concerned, all canines once treated in this way showed dead pulps years later. I applied, therefore, after having gained this knowledge, only light elastics stamped out of rubberdam as the elongating force; these were used only at night twice a week. Generally I preferred merely to make room and leave the tooth alone, if its position was such that it would move naturally in the space created. Certainly natural eruption needs much more time than artifical elongation. The clinical picture resembles a periodontal inflammation with the characteristic signs of looseness and soreness though each of these symptoms may prevail in different cases. All these acute symptoms subside in a short time when the active force is discontinued.

REFERENCES

1. Euler-Mayer: Pathohistologie der Zähne, München, 1927, Bergman.
2. Gottlieb-Orban: Die Veränderungen der Gewebe bei übermässiger Beanspruchung der Zähne, Leipzig, 1931, Thieme.
3. Oppenheim, A.: Bone Changes During Tooth Movement, INT. J. ORTHODONTIA **16:** 535, 1930.
   (a) Tissue Changes Particularly of the Bone Incident to Tooth Movement, Am. Orthodontist, 1911, 1912.
   (b) Biologic Orthodontic Therapy and Reality, Angle Orthodontist **5:** No. 3.4., 1935; **6:** No. 1.2.3., 1936.
4. Orban, B.: Dental Histology and Embryology, Ed. 2, Philadelphia, Blakiston.
5. Marshall, I. A.: A Study of Bone and Tooth Changes Incident to Experimental Tooth Movement and Its Application to Orthodontic Practice, INT. J. ORTHODONTIA **19:** 1, 1933.

THE DENTI-PHORE

A Method for Making Oriented Casts and Recording Dentofacial Changes

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INTRODUCTION

IT IS the purpose of this presentation to describe a practical method for the making of oriented plaster casts and for recording dentofacial changes.

A cast made with this technique is related to four planes of the human head. These planes are: 1. The Frankfort horizontal plane. 2. The median sagittal plane. 3. The auricular plane. 4. The orbital plane.

Following upon the works of Simon, Schwartz, Dreyfus, and others, the underlying principle of the method is as follows: Measurements taken on the head with the denti-phore are transferred to a platform upon which the model is built. These measurements are automatically copied on the base of the plaster cast.

The important new feature of this technique is that the impression is taken as a separate operation, independent of the adjustment of any instruments on the face. This enables the operator to use any method of impression taking and any impression material, including plaster.

INSTRUMENTS AND METHOD

The technique may be divided into two parts—the work on the patient and the work in the laboratory.

The work on the patient consists of the taking of the impressions and the adjustment of the denti-phore.

The denti-phore consists of a U-shaped bar with a horizontal slot in front (Fig. 1). Attached to it by means of a thumbscrew is a mouthpiece with its stem sliding through the slot. Sliding on the U bar are four pointers which can be fixed in any position by means of setscrews.

How to Use It.—This instrument is adjusted to the patient's face in the following manner. The orbital and tragial points are marked on the face of the patient. Some bite wax is softened and rolled into a thin roll, which is then placed on the forked out part of the mouthpiece of the denti-phore. The mouthpiece is then placed in the patient's mouth (Fig. 2), and the patient is asked to bite into the wax until the metal mouthpiece is held rigidly by the teeth. While the patient is holding the mouthpiece, the U-shaped bar of the denti-phore with the pointers upright is guided in position on the stem of the mouthpiece and tightened to it by means of the thumbscrew. While in this position the pointers are adjusted to touch the previously marked orbital and ear points on the patient's face and are firmly secured by tightening the screws (Fig. 3). The denti-phore with the wax bite is then removed and impressions are taken. This completes the work on the patient.

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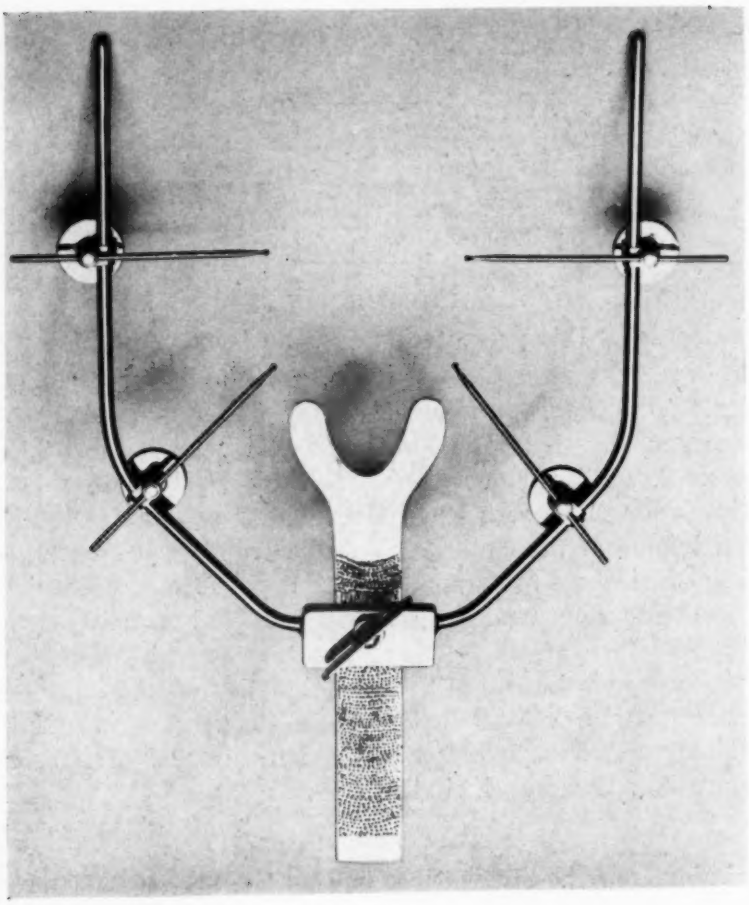

Fig. 1.

Fig. 2.

Fig. 3.

In the laboratory the denti-phore with the wax bite is transferred to the platform on which the model is built. This is done by means of a transfer plate.

The transfer plate (Fig. 4) consists of a plate of steel attached at the center, by means of a ball-and-socket joint, to a metal arm with a square hole which fits on the center post of the platform. This post is graduated in one-half millimeters. The metal arm can be fastened to the post by means of a setscrew. The transfer plate can be set and fastened at any angle to the platform by means of the ball-and-socket joint. Set into each end of the transfer plate is a tubular sleeve, slit through its full length on the outside, where two flanges close the lumen of each tube with a setscrew. These tubular sleeves receive the ends of the U-shaped bar of the denti-phore.

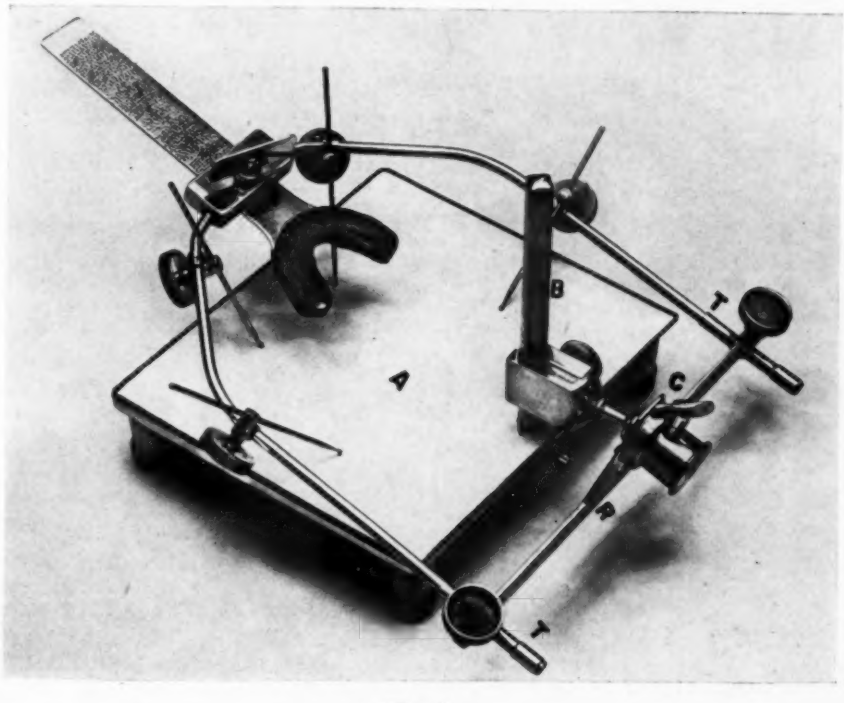

Fig. 4.

The purpose of the transfer plate is to receive the denti-phore and transfer its four pointers to the platform, so that the platform automatically becomes identical with the Frankfort plane. This is done in the following manner (Fig. 4): After the denti-phore with the bite wax has been removed from the patient's face, it is fastened to the transfer plate by inserting the ends of the U-shaped bar into the tubular sleeves and tightening the setscrews. By loosening the ball-and-socket joint and by raising or lowering the transfer plate, by means of the arm, three out of the four pointers on the denti-phore can always be made to touch the platform. When in this position the transfer plate is tightened to the center post and the ball-and-socket joint is made rigid. At this time, a reading is taken on the graduated center post, to register the distance of the arm of the transfer plate from the platform. This reading registers the distance of the occlusal surfaces of the teeth from the Frankfort plane. The platform when touching these pointers is thus made to coincide with the Frankfort plane of the patient. The teeth of the upper model bear the same relationship to the surface of the platform as the teeth of the patient bear to the Frank-

fort plane. The four points of the denti-phore can be transferred to paper to form the Frankfort quadrilateral.

To construct the Frankfort quadrilateral take two sheets of tracing paper slightly smaller than the platform, sheet No. 1 and sheet No. 2. The denti-phore is raised and sheet No. 1 is taped to the platform; a sheet of carbon paper is placed over it and the denti-phore is lowered until the two auricular and the left orbital points touch the sheet. This will mark the points with visible trace on the sheet of paper. Sheet No. 1 is removed and sheet No. 2 is taped. The denti-phore is lowered again until the two auricular and the right orbital points touch and mark the sheet. Place sheet No. 1 over sheet No. 2 so that the auricular points coincide and trace the right orbital point. Sheet No. 1 has the four points. On this paper, the points are united to form the Frankfort quadrilateral (Fig. 5). On this quadrilateral a parallel is drawn to the auricular plane through the left orbital point. A plane drawn through this parallel is the assumed orbital plane. Therefore, the distance from the auricular plane to the orbital plane just obtained is always the distance O—o_1 on the quadrilateral. It is this assumed orbital plane that is now replacing the orbital plane of Simon in the making of our models and our diagrams. Measurements taken from this quadrilateral are used in the construction of diagrams. This quadrilateral is filed away as a permanent record for later use.

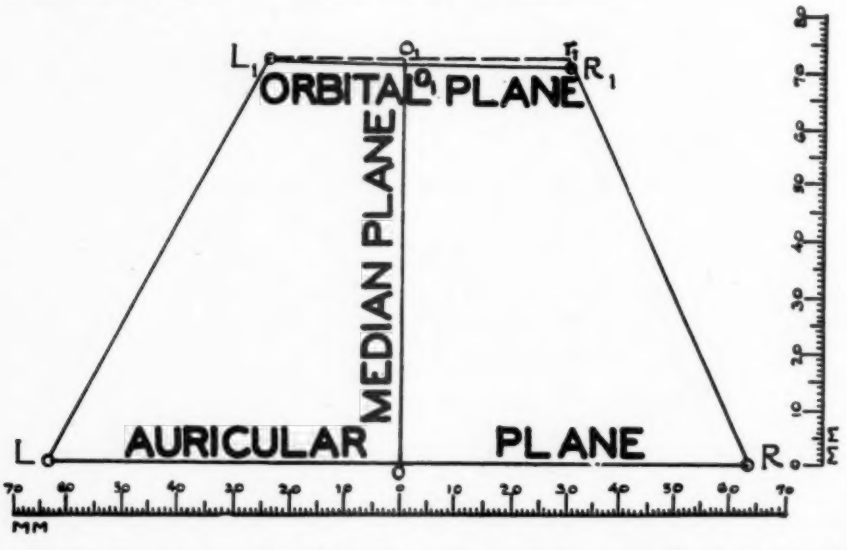

Fig. 5.—The Frankfort quadrilateral. By means of $R_1 L_1 r_1$ we will get the necessary corrections to be applied to the values obtained by using quadrilateral $L L_1 r_1 R$ instead of the true quadrilateral $L L_1 R_1 R$.

Marking the Line Where the Orbital Plane Meets the Frankfort Plane.—The denti-phore is lowered so that the pointers again touch the platform. Where the two orbital pointers meet the platform, two points are marked with a black China-marking pencil. The transfer plate carrying the denti-phore and model is now removed from the square post. With the China-marking pencil, a line uniting the previously marked orbital points is drawn on the platform. This line is the intersection of the orbital and Frankfort planes (Fig. 6).

Marking the Auricular Plane.—The transfer plate carrying the denti-phore and wax bite is replaced on the platform with the orbital pointers falling on the

previously marked orbital line. Where the two tragial pointers meet the platform a line is drawn in the following manner: A ruler with equal graduations on opposite sides is placed against the two tragial pointers (Fig. 6). The distance between the two pointers and the middle of this distance are marked off on the platform on the opposite side of the ruler. The denti-phore is removed from the stand, the ruler is replaced on the platform in the original position, and the three points are transferred to the opposite side of the ruler. With a China-marking pencil, a line uniting the two tragial points is drawn. This line is the intersection of the auricular and Frankfort planes.

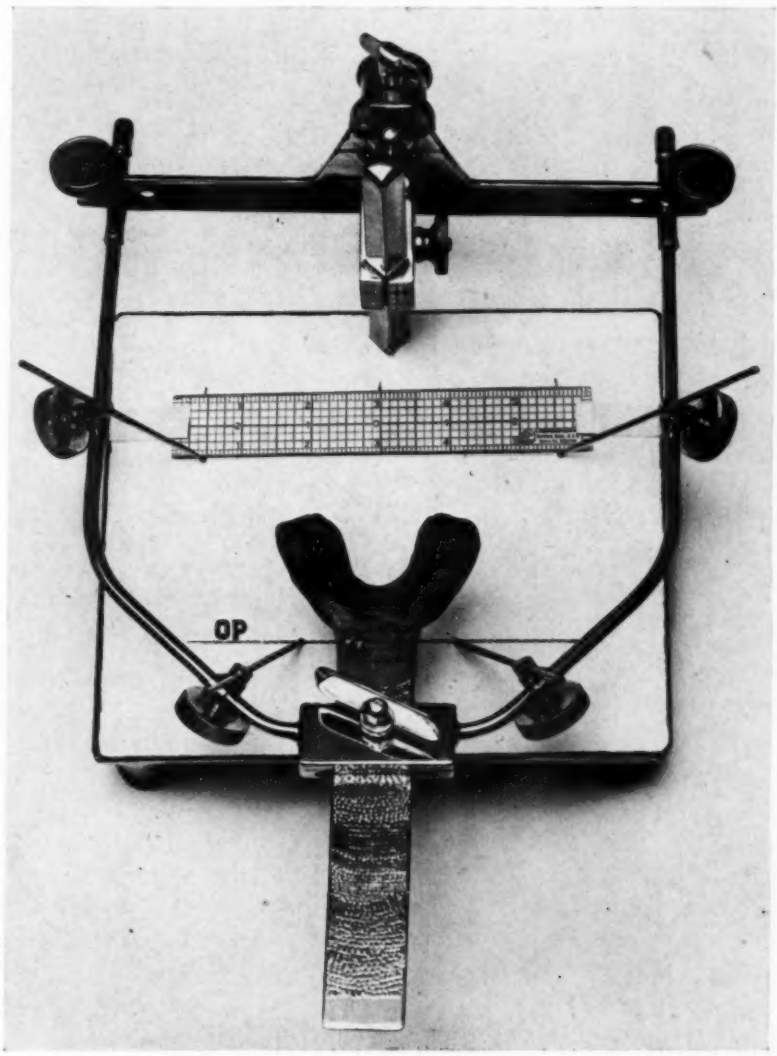

Fig. 6.—*OP*, orbital plane.

Marking the Median Plane.—By uniting the two mid-points and continuing the line across the auricular and orbital lines, a perpendicular to the auricular plane is drawn. This perpendicular will intersect the orbital plane and mark the median line of the head. This median line will not always coincide with the median raphe plane (Fig. 7).

Making the Plaster Base.—After the lines are marked on the platform, the transfer plate and denti-phore are removed from the center post. The pointers are turned up. The upper cast is placed onto the wax bite on the bite fork and secured to it by means of sticky wax and paper clips (Fig. 8). This cast without the base is prepared in the following manner: From upper and lower impressions, which are taken as a separate operation, casts are poured in plaster

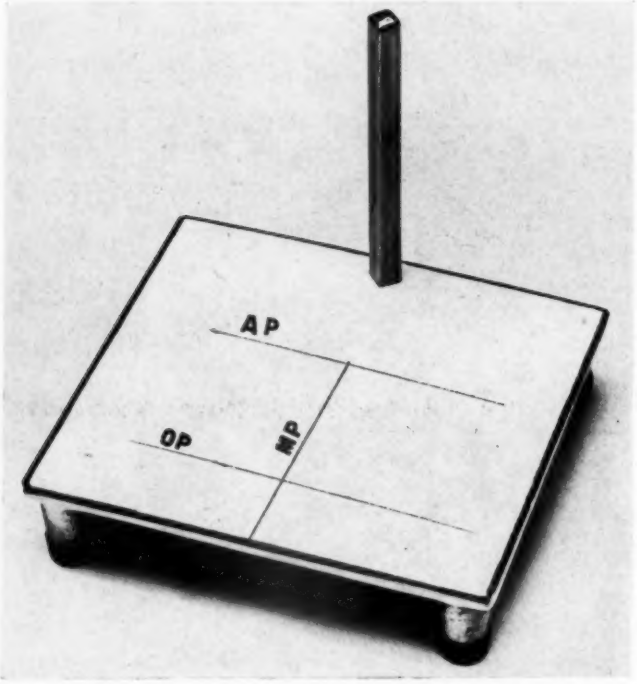

Fig. 7.—*AP*, auricular plane. *OP*, orbital plane; *MP*, median plane.

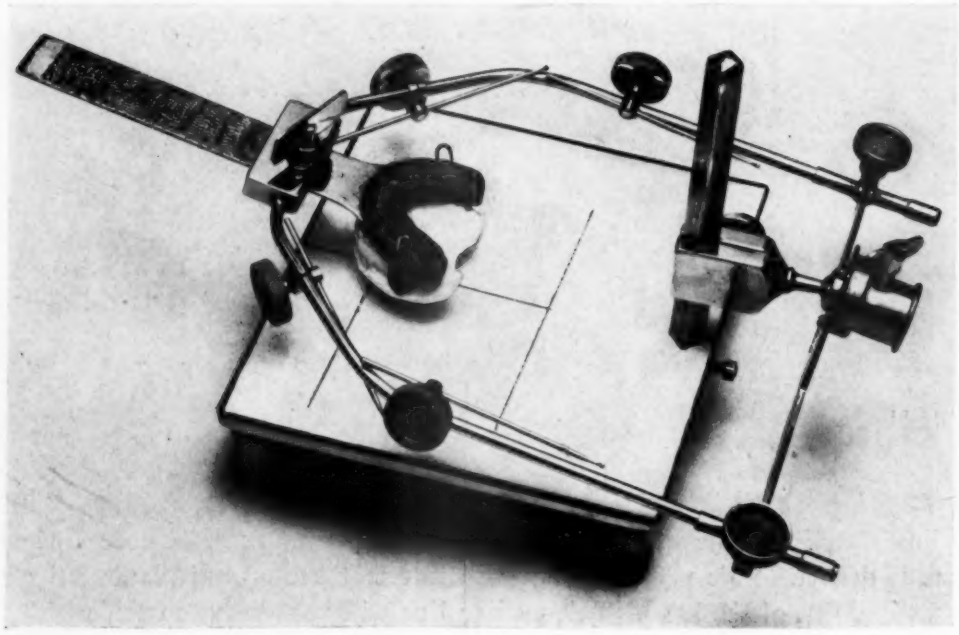

Fig. 8.

or stone and are filled with very little excess over the anatomic portion. After the casts are separated and obtained, the portion just below the teeth is protected by means of adhesive tape. After fastening the upper cast to the wax bite a plaster mix is made and piled on the platform spreading the first portion of the plaster over the marked lines. The denti-phore carrying the cast is then replaced on the square post and guided down until the cast is set into the soft

Fig. 9.

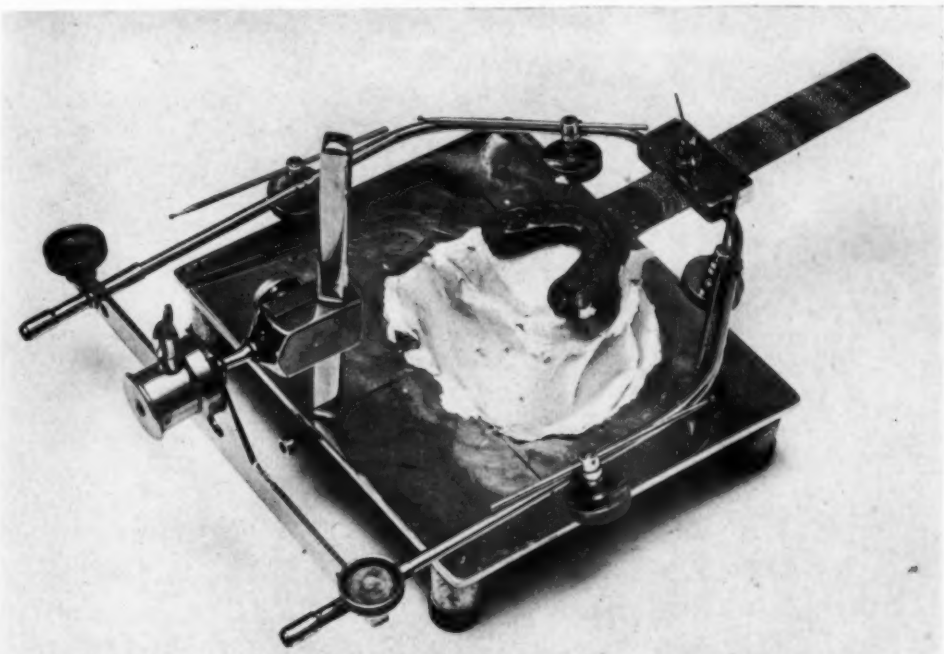

Fig. 10.

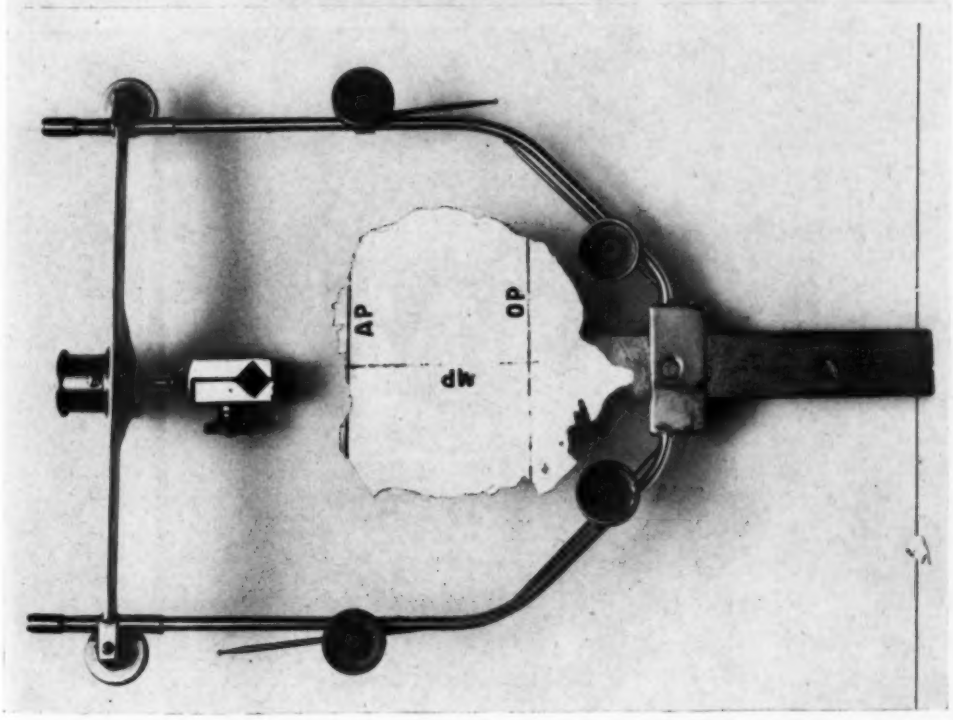

Fig. 10.—*OP*, orbital plane. *MP*, median plane. *AP*, auricular plane.

plaster on the platform (Fig. 9). The reduction in the height of the cast can be decided upon in advance and by tightening the setscrew, the transfer plate is fastened to the square post at any distance from the base. The reduction in height is obtained from the difference in the readings on the square post, taken before and after the turning of the pointers. This difference is recorded and is used to obtain the original height for the drawing of the curves. The plaster is then worked around the cast and allowed to set. The previously marked lines

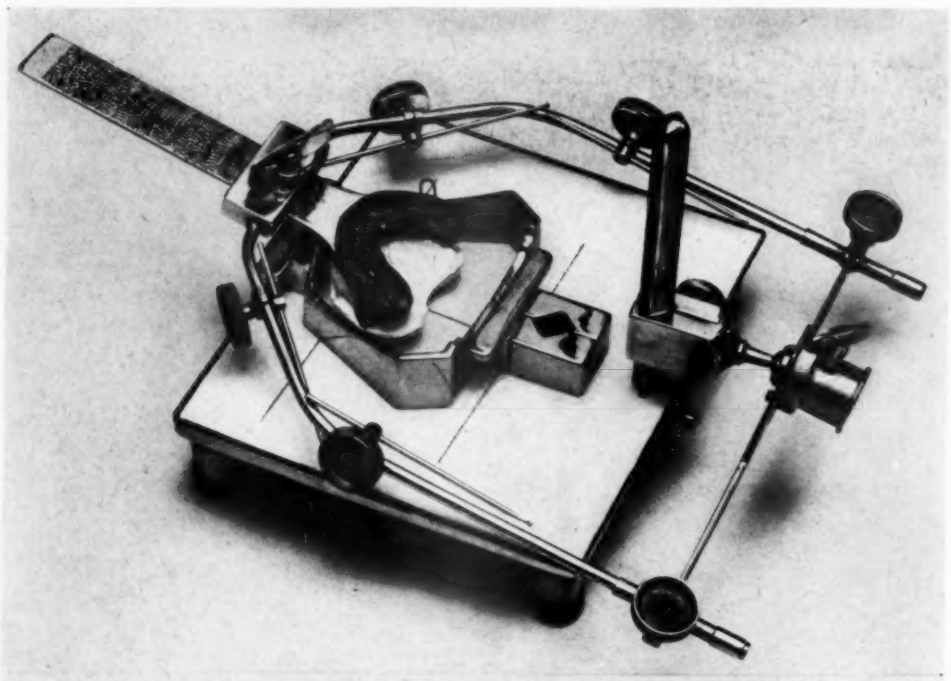

Fig. 11.

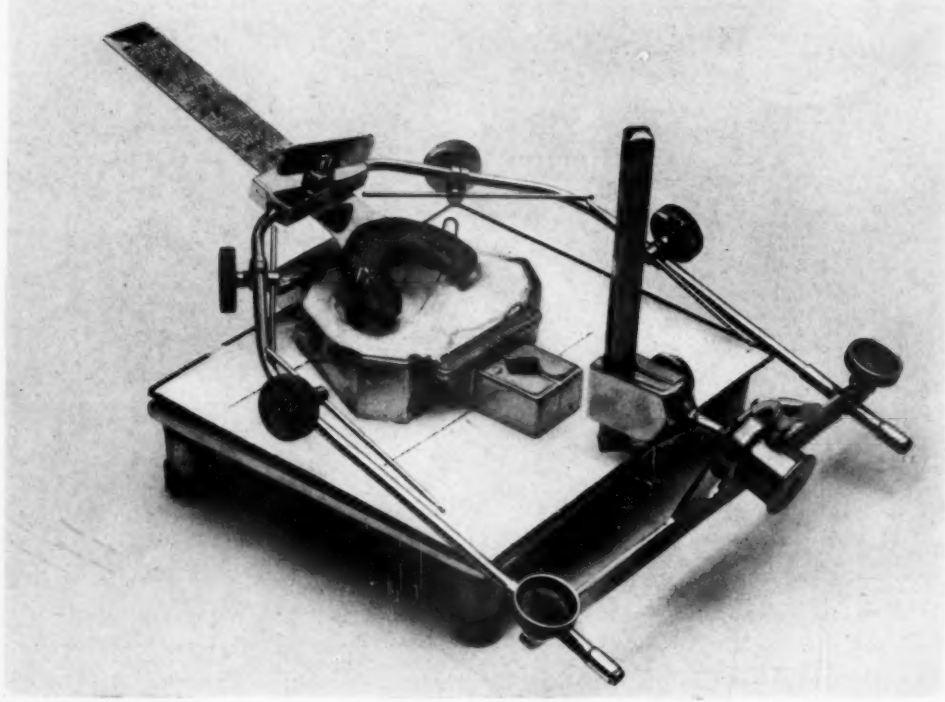

Fig. 12.

on the platform are automatically transferred to the base of the cast (Fig. 10). This makes the base of the cast identical with the Frankfort plane, and on this base is a line representing the orbital plane, another representing the median plane, and still another representing the auricular plane. The sides of the cast are trimmed by hand.

Rings may be used as a preliminary stage in shaping the casts and getting their bases parallel and the final trimming finished by hand. When the rings are used, the ring of the upper cast is placed on the platform (Fig. 11) with its split median line coinciding with the marked mid-auricular line and filled with soft plaster. The transfer plate carrying the denti-phore with the cast fastened to it is replaced on the square post and guided down until the cast is set onto the soft plaster in the ring (Fig. 12). The transfer plate is fastened to the square post at the predetermined height. After the plaster has hardened, the wax is worked away from the cast, and the transfer plate and denti-phore are removed from the stand.

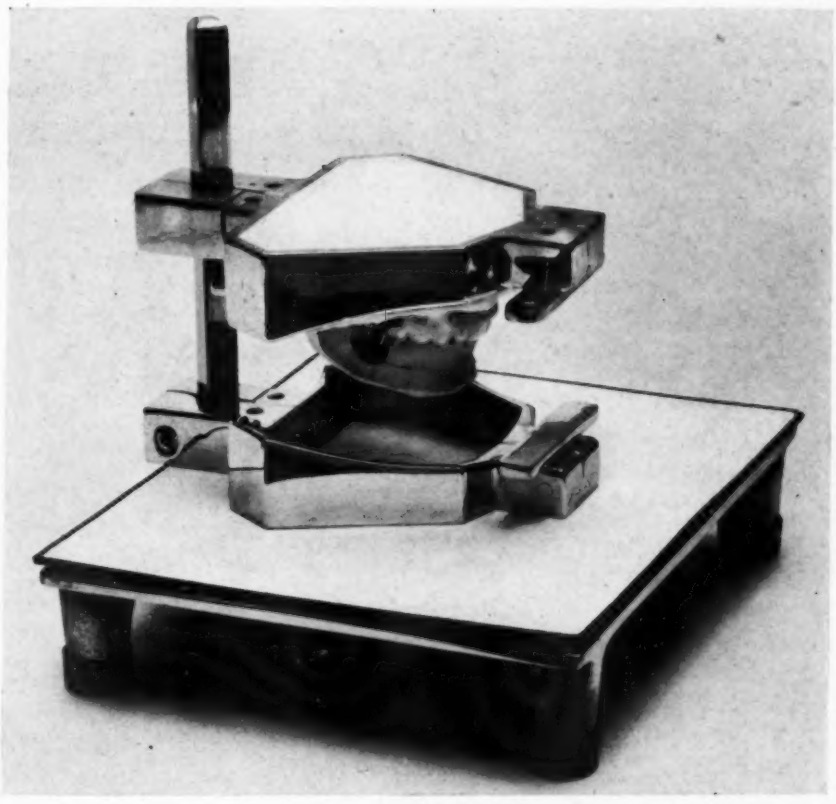

Fig. 13.

The lower cast is completed in the following manner: The lower cast without the base is now occluded with the upper cast, and the two are held together with sticky wax (Fig. 13). In order to prevent the sticky wax from staining the cast, it is advisable not to remove the adhesive tape from the upper cast until the lower cast is completed, this allows the placing of the sticky wax on the adhesive tape instead of on the plaster. The height of the lower cast is decided upon and added to the known height of the upper. The combined height is then

noted on the graduated post. The lower ring is guided down the square post onto the platform and filled with plaster. The upper ring with its cast and lower cast attached to it is carried onto the square post with the lower cast downward. This is then lowered into the soft plaster until the upper surface of the upper cast reaches the predetermined reading on the graduated post. At this position the upper ring is tightened to the bar, and this prevents the lower cast from sinking any further into the plaster than desired. Any additions or smoothing of plaster around the lower cast can now be made, the cast being accessible from all sides. After the plaster has set, the casts are released by loosening the screws. The sides are then finished off by hand to correspond to the lines transferred to the casts.

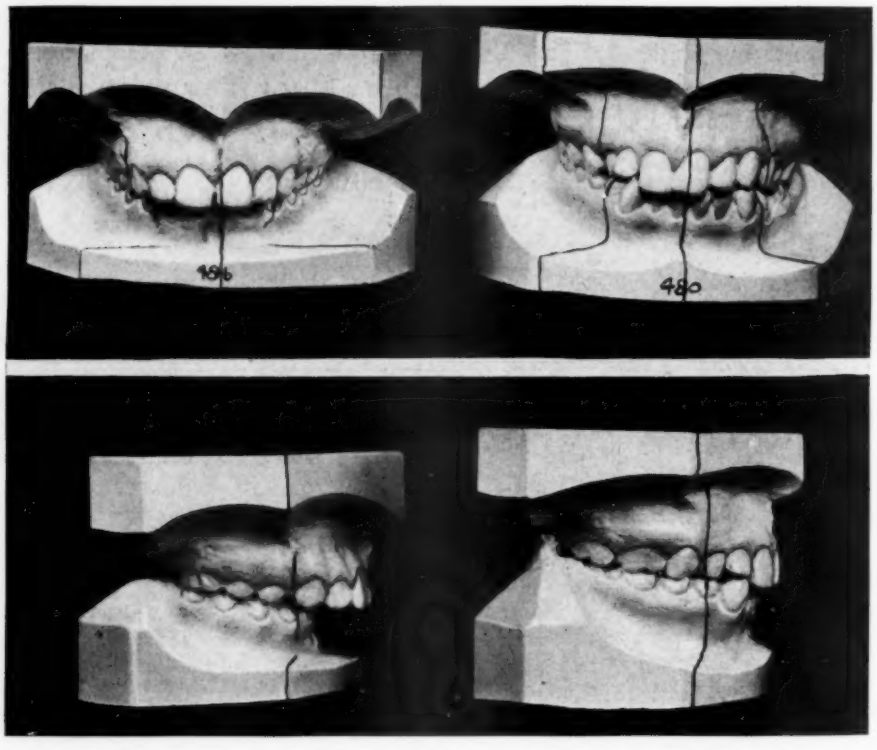

Fig. 14.

RESULTS

Some of the finished casts are shown in Fig. 14.

The value of oriented casts as records in treatment is first fully brought out when we use curves or diagrams made from these casts. By means of such diagrams it is possible to compare dentofacial changes brought about by development or treatment. The occlusal diagram shown in Fig. 15 was made from casts of a patient referred to me for continuance of treatment. The only records we had before treatment was started were the gnathostatic casts. After the completion of treatment, a slanting down of the occlusal plane from left to right was noticed. The question arose whether this asymmetry, which was quite marked, was produced during treatment or whether it was present before treatment was started. An inspection of the diagrams (Fig. 15) made from these casts shows that the right and left angles formed by the Frankfort plane and a

tangent to the occlusal curve are the same for both casts. This is expressed in the distance "Y" which is nearly the same for both; 439 and 439A. (439 is before treatment, and 439A is after treatment.) This proves that the asymmetry of the occlusal plane was present before treatment was started.

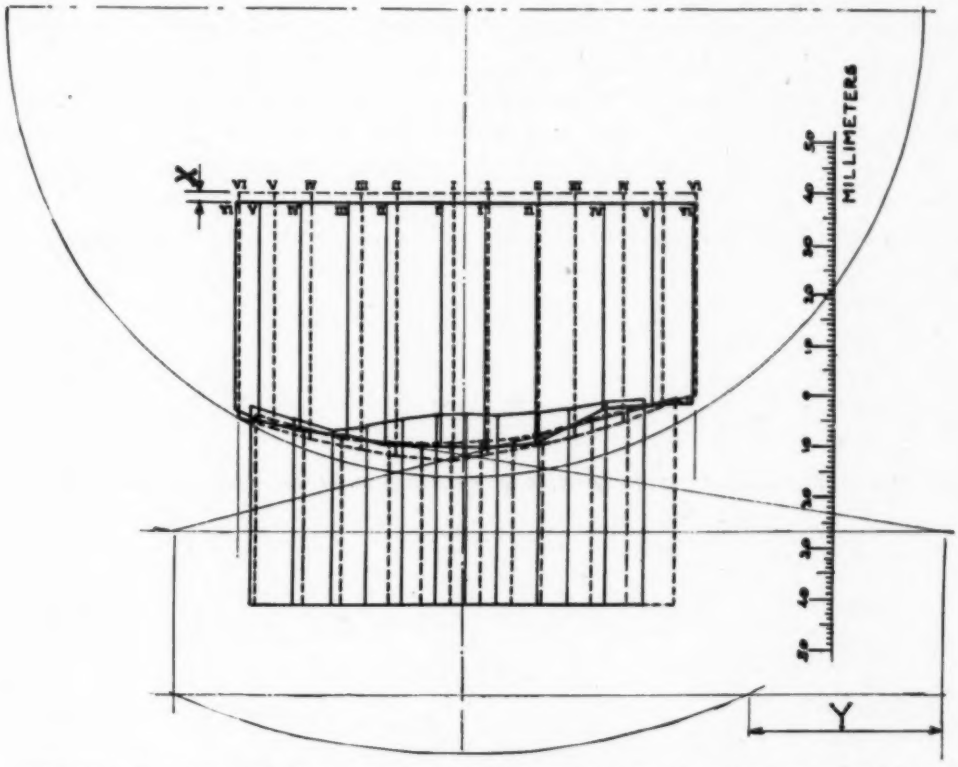

Fig. 15.—Superimposed diagrams showing distances of teeth from the eye-ear plane before and after treatment. All values used in the diagrams were obtained from oriented casts made before and after treatment. Solid lines, No. 439 before treatment. Broken lines, No. 439A after treatment. *X*, distance in millimeters representing reduction in overbite after treatment. Distance *Y* which is nearly the same for both 439 and 439A shows the horizontal asymmetry of the left side of the occlusal curve was present before treatment.

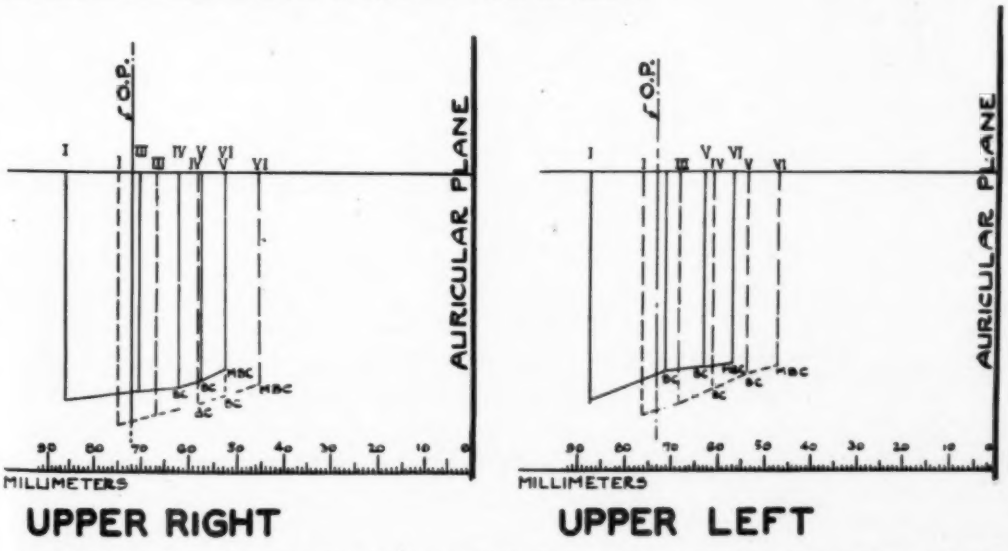

Fig. 16.—Diagrams showing distances of teeth from the auricular plane before and after treatment. All values used in diagrams were obtained from oriented casts made before and after treatment. Solid lines, before treatment, No. 439. Broken lines, after treatment, No. 439A.

A very useful diagram is the sagittal diagram shown in Fig. 16. This diagram shows the distances of the teeth from the auricular plane before and after treatment. In this diagram, there are three planes: the Frankfort plane; the orbital plane; and the auricular plane. The Frankfort plane is the same plane as used by Simon. The orbital plane is an assumed plane and is not identical with Simon's orbital plane. The auricular plane passes through the ear points and is perpendicular to the Frankfort plane.

To construct the sagittal diagram lay off distance O—o_1 obtained from the quadrilateral (Fig. 5) on a line representing the Frankfort plane. At the end of the measured distance construct a perpendicular to the Frankfort plane. This plane is the auricular plane on the sagittal diagram. At the other end of the distance construct another perpendicular which represents the assumed orbital plane. The distances of the teeth from the auricular plane are then laid off on the Frankfort plane.

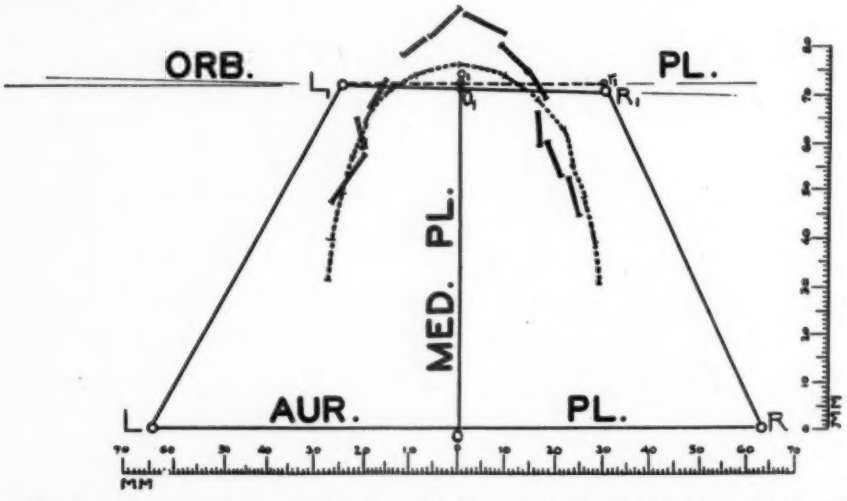

Fig. 17.—Curves representing projection of teeth on the Frankfort plane with reference to the auricular and median planes before and after treatment. Solid lines, before treatment. Dotted lines, after treatment. All teeth shown in this curve were located by their distances from *O* o_1 as abscissae and by their distances from L_1 r_1 as ordinates. The distances of the teeth from *L R* are obtained by subtracting from *O* o_1 their respective distances from L_1 r_1.

In this diagram either the orbital or auricular plane can be used to show changes in the mesiodistal position of teeth. However, in the interpretation of the final diagram the following possibilities must be considered:

1. That the plane has remained fixed, the teeth therefore have been moved back.

2. That the teeth have remained in a relatively fixed anteroposterior position, the plane therefore has moved forward in the process of growth.

3. That neither the plane nor the teeth have remained fixed; the results therefore must be partly attributed to a forward movement of the orbital plane through the natural development of the face and a distal movement of the teeth through treatment. We know that there is no absolutely fixed point in the head but this much could be said in favor of the auricular plane: being located in the center of the head should make it relatively more fixed than any plane anterior to it.

NOTE: It must not be overlooked that the change in the mesiodistal position of the teeth is partly due to the widening of the arch in treatment.

Another form of diagram is shown in Fig. 17. This diagram shows at a glance the position of the teeth before and after treatment in relation to the median, the orbital, and auricular planes.

Some thought has been given to the possibility of correlating the planes registered on the sagittal diagrams made from these oriented casts with corresponding planes on facial photographs. This correlation appears to be possible if the planes are registered directly on the negative at the time the photographs are taken.* These photographs must be taken at the same sitting when the denti-phore is adjusted to the face. A tracing of such a *profile* photograph with superimposed sagittal diagram is shown in Fig. 18. Definite work on this has not been completed.

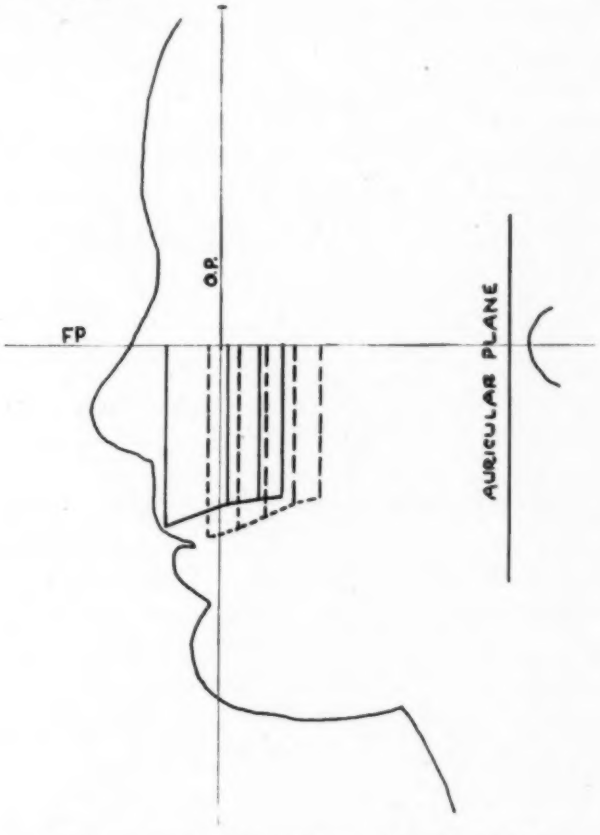

Fig. 18.—*FP*, Frankfort plane. *OP*, orbital plane.

SUMMARY

In summing up, the following essential points of the apparatus and method may be stated:

1. The denti-phore makes it possible to take the impression as a separate unit.

2. The transfer plate automatically registers the measurements of the face on the base of the cast.

3. Time consumed on the patient and in the making of casts is reduced to a minimum.

*Such photographs are obtained with the Cephalo-Phore. The method is fully described in AM. J. ORTHODONTICS 26: 2, 1940.

4. Advantages of an oriented cast obtained by this technique are:
   a, From the base or Frankfort plane changes in vertical positions of teeth can be recorded. In changing the occlusal plane through treatment the oriented cast will show whether this was accomplished by depressing certain teeth, elongating other teeth, or both.
   b, Measuring from the same plane, changes in overbite can be noted.
   c, Changes in the axial position of teeth can be noted from a comparison of the long axis of the teeth with the parallel sides of the cast and their relation to the Frankfort plane.
   d, Mesiodistal movement of the individual teeth or segments of the arch cannot be ascertained from the orbital plane alone on the oriented cast. In this connection the value of having an additional plane must not be overlooked.

REFERENCES

1. Simon, Paul W.: Fundamental Principles of a Systematic Diagnosis of Dental Anomalies, Boston, 1926, The Stratford Co.
2. Porter, Lowrie J.: Orthodontic Plaster Model Former, First Int. Ortho. Congress, St. Louis, 1927, The C. V. Mosby Co.
3. Schwartz, Rudolph: Cephalometric Methods and Orthodontia, First Int. Ortho. Congress, St. Louis, 1927, The C. V. Mosby Co.
4. Andresen, Viggo: Die Gnathophormethode. Die Künstlerische Diagnose und wissenshaftliche gnathophysiognomische diagnose, Fort. d. Ortho., 1936, Leipzig, Hermann Meusser.
5. Schray, Karl: Die Klärung der Kiefer-Gesichtsbeziehungen bei Bissanomalien, Fort. d. Ortho., 1935, Leipzig, Hermann Meusser.
6. Schüssler, Helmut: Eine neue Methode der odontorthopädischen Kiefermessungen, Fort. d. Ortho., 1935, Leipzig, Hermann Meusser.
7. Dreyfus, Sylvain: The Taking of Impressions and the Automatic Casting of Models for Orthodontic Diagnosis, INT. J. ORTHODONTIA **19**: 2, 1933.
8. Fischer, Bercu: Orthodontic Model Former and Face Bow, AM. J. ORTHODONTICS **24**: 6, 1938.
9. Idem: Orthodontic Photographic Recorder, AM. J. ORTHODONTICS **26**: 2, 1940.
10. Martin, R.: Lehrbuch der anthropologie, Jena, 1928, G. Fischer.

2 EAST 54TH STREET

CONSTITUTIONAL DISEASES IN THE PROGNOSIS OF ORTHODONTIC TREATMENT

The Necessity and Usefulness That Orthodontics and Dentistry Re-Enter the Field of General Medicine

Prof. Dr. C. D'Alise, Naples, Italy

WHEN Dr. Lockett, at the First International Congress of Orthodontia, held at New York in 1926, dared to call attention to the grave problem of the frequency of relapse in orthodontic treatment, it seemed to many that he had brought a discordant note into that first international gathering, in which orthodontics showed its scientific maturity and its great progress.

Since then thirteen years have passed, and the problem enunciated by Lockett still remains grave and unsolved.

It is true that in these last years the problem of the failures in orthodontic treatment has been very opportunely brought to the stage of discussion, but the fundamental problem regarding the failures and relapses in orthodontics, however universally recognized and admitted by the most expert students of our specialty, has not yet determined a new direction, that looking at the past and profiting by all the progress of physiopathology of the dental system, and above all of the etiology of the dentofacial deformities, brought to a clinical constitutional diagnosis, and consequently to a rational and sane prognosis and therapeutic.

The study of the causes of dentofacial deformities, in fact, has shown clearly, and in an undiscussable mode, that there exist close and important relations of interdependence between the physiopathology of the mouth and that of the other organs of the body. Therefore, it is not possible to study and know the physiopathology of the mouth without studying and knowing that of the rest of the body, and so much less is it possible to pretend to institute rational treatment of dentofacial deformities, excluding the organico-constitutional state of the patient.

It has often been said that orthodontics, in these last years, has passed from the purely technical mechanical field into the biologic. How can one speak of biology in dentistry and orthodontics, if these specialties still live autonomously; that is, separated from general medicine, of which they are integral parts?

Biology is the science of life, and the life of the human body is maintained by the functional harmonic collaboration and interdependence of all the organs.

It is true that the other specialties of medicine also follow an analytic direction without synthesis, but the others' error, if it attenuates ours, does not justify it.

The progress of dentistry and orthodontics in these last fifty years has been truly great, but such progress has not been completely valued, even by those

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who have produced it. This is because they have not considered that the human organism is a harmonic, organic, constitutional whole, which must be studied in its anatomic, physiopathologic, and clinical entity, without which every specialty may progress in the technique of etiologic and curative research of diseases, but sterilizes itself in the biologic and clinical field.

Speaking then of biology of the human organism, or of a part of it, without knowing all the others, is nonsense, which can produce but illusions.

Now, turning to orthodontics, why do we wonder that there are frequently some failures and relapses in orthodontic treatment? That perhaps the orthodontic treatments are not subject to the same biopathologic laws that govern the treatments of diseases of other parts of the body? The frequency instead of the failures and relapses should call us to the sad reality of the facts, to the deficient knowledge we have of etiopathogenesis of the dentofacial deformities.

It is not that we are wanting in the studies and researches in this field; rather they have been many and very important, but they have not been conveniently valued because we are lacking in the synthetic criterion of the human organism, synthetic criterion that, besides, lacking also in the students of the other specialties of medicine and surgery, for we note that in spite of so much progress made in medical science, it is not alone the dentofacial deformities that increase, in number and gravity, but it is the whole human organism that declines.

The cause of all this depends on the facts that:

1. On one side, physicians have not yet been convinced that it is not possible to progress in the knowledge of the physiopathology of the human body, ignoring or neglecting the study of the physiopathology of the mouth, of which the dental system is the fundamental organic nucleus.

2. One cannot speak of the functions of digestion and nutrition, excluding from them the integral function of mastication.

3. It is not possible to speak of the normal organic development of the human body, and especially of the face and digestive tract, singling out from it the normal development of the dental system and the mouth; while, on the other hand, dentists and orthodontists are obstinate in an autonomism that does not let them see that dentistry and orthodontics cannot progress farther if they do not re-enter the field of general medicine.

Of the necessity that dentistry and orthodontics re-enter in general medicine with integrative function, I have spoken in my several works and more especially in that published for the First International Congress of Orthodontia,[1] and more recently in that given at the Tenth French Congress of Stomatology,[2] and here, speaking of the constitutional diseases in the prognosis of orthodontic treatment, I propose to demonstrate that at the actual state all the specialties of medicine and surgery, dentistry and orthodontics comprised, if they would not sterilize themselves in a deleterious autonomism, must all draw their life from general medicine as the branches from the vine.

Prognosis in orthodontics is rarely spoken of and only in these later times some author begins to hint of it. Yet prognosis is the most difficult part in the

treatment of diseases because it represents the synthesis of the anatomic, physiopathologic, and clinical knowledge of anyone who prepares himself to treat any disease.

Prognosis is the judgment in advance concerning the duration, course, and termination of a disease. It, therefore, owing to the multiplicity of facts of which it demands the knowledge, and of which a great part escapes the preventive appreciation, is very easily subject to error, is always difficult, so that in the majority of cases one can only give a statement of relative certainty, if not even of simple probability.

The first condition for a prognosis of relative certainty in every disease is an exact etiologic diagnosis, because, as is known, the causal treatment must be our first task.

A clinician, A. Murri, states: "Even after having made the generic diagnosis of typhoid fever or of another disease, you must make a special diagnosis of your patient day by day."

Now if in the acute infective disease, in which the determined cause may be easily ascertained, the prognosis is always reserved and depends in great part on the organico-constitutional conditions of the patient, which establish the severity of the disease and determine the course most favorable, in the prognosis of the dentofacial deformities, in which the etiology is generally complex and the treatment long, we must keep account of the constitutional state of the patient.

In fact, to establish the approximate diagnosis of dentofacial deformities, to make a prognosis of probability, we must consider, besides the degree of the deformity, the patient's age, the hereditary history, and above all, the constitutional state of the patient, since it is obvious that the lighter the degree of the deformity, the more favorable will be the result of the cure. Therefore, the younger the patient, the easier it is to correct the deformity; thus, under equal conditions an acquired deformity is more easily corrected than a hereditary deformity. The course of the treatment depends, in great part, on the way the organism answers to the stimulus that we apply to the alveolar bone tissue through the teeth, and this way of answering to the curative treatment is in close keeping with the constitutional state of the patient, not only at the time of the examination, but also in developments during the course of the treatment, through intercurrent diseases that may occur.

Now, if we consider that constitutional diseases today are in continuous increase, we can also explain the reason why even in expert hands, the failures are not rare, but relatively frequent.

For my part I can affirm that in almost thirty years of orthodontic practice I have noted that in these last years, in spite of increased experience and improved curative technique, the organisms in general respond less well to the curative treatment than when I was much less experienced. In certain cases even the minimum stimulus, and I say almost insignificant, that I apply to the teeth for their displacement is followed by a phenomenon of intolerance of the alveolar bone tissue, and more especially by the apical base, almost as if it were an inert tissue.

In these cases I survey the reaction of the bone tissue almost day by day and do not fail to submit patients to a general reconstituent cure, constituted

by administration of cod liver oil, calcium preparation, vitamin, opotherapic preparation, etc., by my initiative, or more often in accord with the patient's physician, but with very poor results.

And I do not blame my curative method for these poor results of the cure I practice. For in a general line for twenty years I have used the ribbon arch, modified in its metallic alloy so as to render it ductile like lead, and so as to allow for the slightest movements, and have found that patients of good constitution achieve excellent results.

I am convinced that the constitution of civilized man today is getting continually worse, and, therefore, we note a greater morbidity, a greater frequency of general diseases, and dental diseases especially. It is not to be marvelled then if the failures in orthodontic treatment, as I have before said, are frequent even in expert hands.

Regarding the relative frequency of the relapses, to my mind it depends on the fact that in orthodontic cures we often practice the symptomatic cure and not the causal. I infer from this that, in the etiologic study of the dentofacial deformities, we do not take into due account one of the fundamental causes and that is of the deficient function of the dental apparatus since its first appearance in the infant's mouth.

That the vigorous and integral masticatory function has a capital importance in the normal development of the jaws and the face is an undeniable fact. It is undoubtedly the merit of dentists and orthodontists who have demonstrated and clinically and experimentally proved this very important functional finality of the dental system. This is also shown by the efficiency of the myofunctional therapy, in the correction of dentofacial deformities, studied and applied by Rogers and others.

It is true that the myofunctional therapy is not directly concerned with mastication, but it is one of its surrogates and tends to the functional reeducation of the muscles of the face which are also involved in mastication.

However, to prevent the relapses of our patients, we must put in practice the masticatory function in its natural integrity and activity, bearing in mind that with modern alimentation of the civilized peoples, whose aliments are nearly always soft, the mastication they require is the parody of true and natural mastication.

That dentofacial deformities have always existed is an incontestable fact, but that they, in the present generations of the civilized peoples, are in continuous and progressive increase in frequency and gravity is also proved today by accurate research of anatomists, anthropologists, and orthodontists.

Sir Arthur Keith, the English anthropologist, after historical researches on some series of ancient skulls, states that, "Every fourth or fifth child or adult we examine possesses a palate, which, compared with the older type, may be described as both deformed in shape and reduced in size." On another occasion, commenting on his comparison of pre-Norman skulls with fifty modern skulls, he says, "Among collections of modern skulls we see teeth, jaws, and faces as robustly and symmetrically developed as in ancient times, but they occur in decidedly diminished proportion."

Prof. Brash, the English anatomist, after accurate examination of a collection of sixth century Anglo-Saxon skulls found in Bidford Cemetery says:

"Although the frequency of some recognizable malocclusion is very great in the Bidford collection, yet the average degree of malocclusion is not nearly so great as in modern British people, and it consists to a considerable extent of irregular positions of individual teeth. On the whole, the palates of those Saxons are notably broader than the average modern English palate."

Numerous orthodontists have made similar studies. Among these Dr. Waugh of New York, in a recent study on the Eskimo, shows how in these, following a change of diet, the jaws are deformed in one or two generations, causing every kind of malocclusion, and to prove this he presents models of grandfathers, fathers, nephews, and nephews' sons, while Sir Colyer, an illustrious English stomatologist, in a study on the pathology of the crowded mouth, admits that this condition of the mouth is due to the insufficient development of the jaws that no longer allow a normal disposition of the teeth on the jaws.

Now, given the impressionable number of children affected by dentofacial deformities, which in England, according to Corisande Smith, reaches 90 per cent and in other nations varies from 60 to 75 per cent, we must, before all, ask ourselves what meaning the dentofacial deformities have in the development and life of man.

For the anthropologists and many orthodontists it would be in direct report with evolution, as in the ulterior progress of man the face must diminish in volume to allow for increase in volume of the brain. We cannot admit such an interpretation of decisively pathologic manifestations, and this for a complexity of theoretic and practical reasons:

1. Because so far, in spite of the great number of studies made to demonstrate the exactness of the Darwinian evolution, we have not certain fact to admit it.

2. There exists no relation between the diminution of the face and the increase in the brain.

3. That in Creation all is harmony and proportion, and therefore the true progress of a living organism is always harmonic and proportional, as the inharmony of its parts is always a pathologic fact.

4. That the dentofacial deformities, among other damage, disturb in a greater or minor grade the two fundamental functions of vegetative life, digestion and respiration, and not rarely also the intellect.

Thus, if the dentofacial deformities are, as I have also tried to demonstrate in my recent work,[3] degenerative pathologic manifestations, how can one affirm that man progresses? How can one conciliate the real progress of medical science and the decline of the human organism?

Very often I have called the attention of colleagues—physicians, orthodontists and dentists—to the fact that the analytic tendency without the consequent synthesis in the contemporary medical studies, leads fatally to their sterility. Man, like the rest of every living organism, must be studied and considered as a whole. The study, instead, of his separate parts may be made alone, and to a better understanding of the whole, but not as an end in itself.

Now, how is it possible to understand the whole, if we do not know all the parts? And if we do not know all the parts, how is it possible to conceive that of a living organism; while some of its parts are ill, degenerate, others are better?

Yet many physicians, and not a few stomatologists and orthodontists, have had these conceptions, and still have them!

Vain and damaging illusions of the Darwinian evolutionists who misjudge the degenerative, somatic notes for signs of human progress!

We orthodontists often reproach the anatomists and anthropologists for the want and inexact valuation of forms of malocclusion, but we must also reproach ourselves for the want and inexact valuations of the cause and meaning of them, and their progressive increase.

The diseases of nutrition today, notwithstanding the incontestable progress of the chemistry of alimentation, dominate the field of human pathology. This is due, above all, to the fact that we have misunderstood the very great importance that the mouth has in the preparation and digestion of food.

We must return to the ancient aphorism, *Prima digestio fit in ore,* fruit of human wisdom and not abstract science, to understand that to make the human race, which is in decadence, healthy, it is necessary that dentistry and orthodontics return, with integrative function, into general medicine to re-establish the anatomic, physiopathologic, and clinical harmonic unity of the human body which physicians and dentists have lost sight of.

REFERENCES

1. D'Alise, C.: Dentistry and Orthodontia in Medicine of the Twentieth Century.
2. D'Alise, C.: Le rôle de la stomatologie dans la médecine contemporaine et dans l'avenir.
3. D'Alise, C.: La razza umana è in decadenza. L'aumento continuo e progressivo delle deformità dento-facciali ne è un segno certo, Rivista italiana di Stomatol., February, 1937.

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PLASTIC SURGERY OF THE FACE

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CLEFT LIP AND PALATE

UNTIL recently the surgical treatment of the cleft lip and palate had remained more or less stationary. Although the results with the older types of operation were fairly good, much remained to be accomplished, especially in esthetics, following the lip operation and function following the palate operation.

Many of these unfortunate people have isolated themselves from society because of their appearance or their speech. A few have been able to readjust themselves in spite of their handicap.

Modern harelip and cleft palate surgery carries the prediction of an almost normal appearance and speech.

The Mirault-Blair operation marks a distinct advance in the treatment of single harelip. Not only is a good lip reconstructed but a good nose as well. Older operations concerned themselves with closing the fissure in the lip and leaving the nasal deformity uncorrected. Today, our attention is first directed to the nasal deformity, and rightly so, because the external nose is a more conspicuous feature than the lip. In the male, defects can be partially obscured by growing a mustache.

Although the Mirault-Blair procedure is quite technical, once it is mastered, the results obtained with it are superior to any other method (Figs. 1 and 2).

The correction of bilateral lip clefts leaves much to be desired, Fig. 3. If the clefts are very wide, a tight lip is the usual end result.

Lip clefts, whether single or double, are closed over the open clefts in the jaw.

Ten to fourteen days after birth is the preferable time to close lip clefts.

Secondary corrections in later years are frequently necessary in the majority of cases, Figs. 4 and 5.

In closing the cleft palate, the classic Dieffenbach-von Langenbeck operation has been most widely used. Although it has been possible to obtain a

Fig. 1.—Unilateral harelip. *A*, Before correction. *B*, Two weeks after correction by the Mirault-Blair technique.

Fig. 2.—Incomplete harelip. *A*, Before operation. *B*, Two weeks after operation. The technical procedure is essentially the same for complete or incomplete clefts.

Fig. 3.—Double cleft of lip and jaw. *A*, Before operation. *B*, Six weeks after operation.

good surgical result using the older methods, the functional results have not been good. The proponents of osteal uranoplasty claim better functional results, but, on the whole, we have failed to observe any improvement over the classic operation.

In recent years, the development of the push-back operation has resulted in producing a better functional palate. When the palate is closed by the

4A. 4B. 4C. 4D.

5A. 5B.

Fig. 4.—Secondary harelip deformity. Primary operation on lip at birth. A good lip has been formed but a poorly constructed nose. *A*, Ala droops and is markedly concave. *B*, After correction, by removing an ellipse from the inner surface of the ala. *C* and *D*, Same viewed from below.

Fig. 5.—Secondary harelip deformity. This is probably the most common nasal deformity seen, following poor primary repairs. *A*, There are a marked drooping, flatness, and spreading of the left ala. *B*, Two months after correction. The columella was divided from the septum and lip and sutured in a more advanced position.

usual methods, it is too short to permit nasopharyngeal closure, and this latter is very essential in speech production.

In performing the push-back operation, the entire soft palate is displaced posteriorly, Figs. 6 and 7. Preserving the blood supply through the descending palatine vessels is necessary. Around two years of age is a good time to perform this type of operation.

NOSE

The nose is a very conspicuous facial feature and is subject to a wide variety of deformities.

Nasal deformities are quite common following primary harelip repairs, and have been mentioned above.

The long nose is a distinct racial characteristic, Fig. 8. It is due to an excessive length of the septal and lateral cartilages. The columella may be short and drooping and cause a retraction of the tip of the nose, Fig. 9.

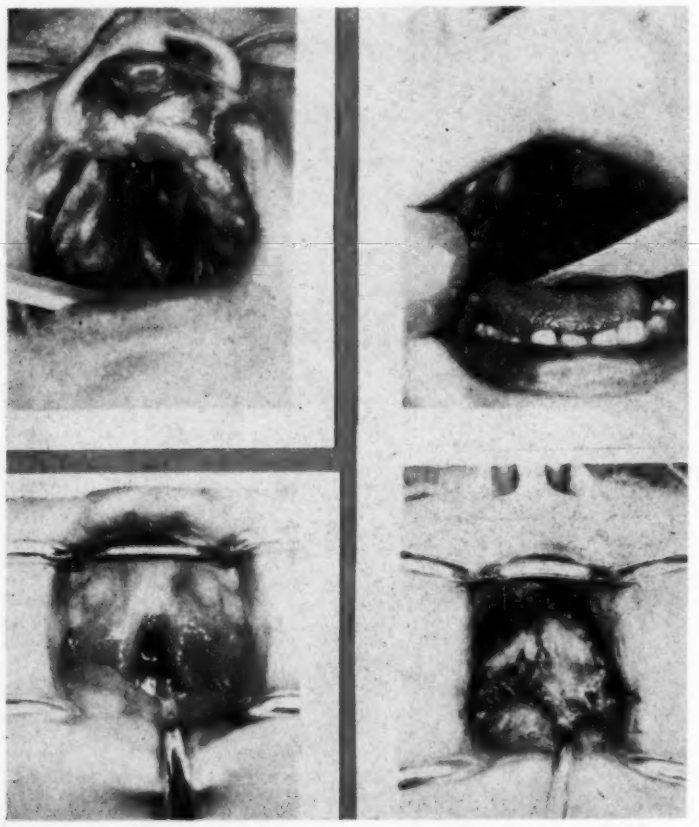

Fig. 6.—Double cleft of the palate. *A*, Before operation. *B*, One month after correction by the push-back method. The palate is sufficiently long to touch the posterior pharyngeal wall and effect nasopharyngeal closure.

Fig. 7.—Cleft of the soft palate. *A*, Before operation. *B*, Immediately after operation. Area of palate left bare of covering after the set-back is shown by the black area. The palate was set back enough to make nasopharyngeal closure possible and at the same time to allow for some contraction which takes place.

Nasal injuries are common and very often are but part of more extensive facial injuries. Swelling masks the injury to a great extent, and if one is not careful in repositioning the fractured parts, deformity is sure to follow, Fig. 10.

EARS

Outstanding ears is a common deformity, Fig. 11. It is due to a failure of complete development of the antihelix. Former operations consisted of excising a section of skin from behind the auricle, but this did not result in a permanent correction. The method of J. S. Davis which reconstructs the antihelix results in a permanent correction. The ears should not be set too

8*A*. 8*B*.

9*A*. 9*B*.

10*A*. 10*B*.

Fig. 8.—Long nose. *A*, Before correction. *B*, After correction by an excision from septal and lateral cartilages.

Fig. 9.—Retraction of tip of nose, drooping and short columella: *A*, Before correction. *B*, After reduction and lengthening of columella and advancement of tip.

Fig. 10.—Deviation of nose, following automobile injury. *A*, Before correction. *B*, After correction. Nose was sawed through the frontal process on either side and shifted to the midline.

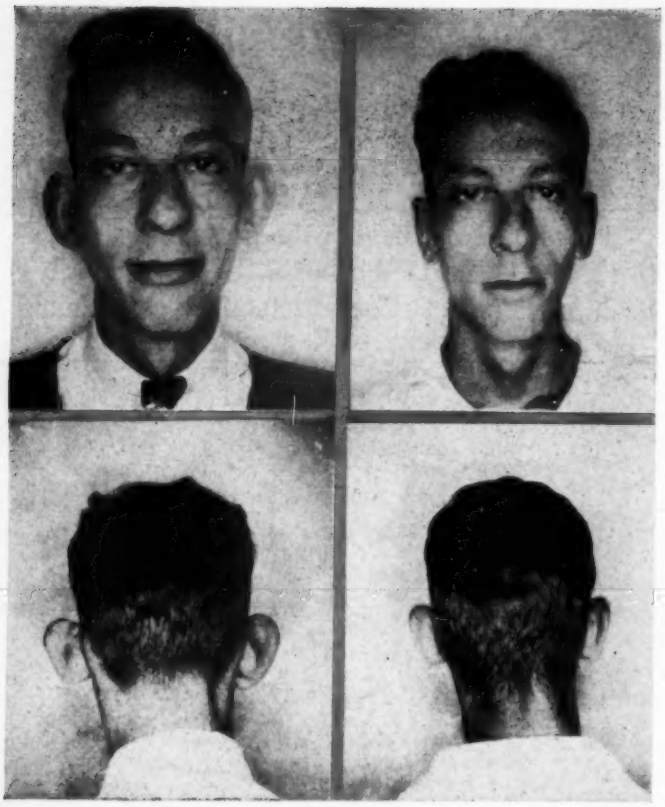

Fig. 11.—Protruding ears. *A* and *B*, Front views, before and after correction. *C* and *D*, Back views, before and after correction.

Fig. 12.—Moderate receding chin. *A*, Before correction. *B*, After insertion of ivory implant.

Fig. 13.—Facial scar, following an automobile accident. *A*, A trap door type of laceration was present which had been poorly repaired at the time of the injury. *B*, Condition six months after secondary repair, by the subcuticular method.

close to the head. A cephaloauricular angle of about thirty degrees will result in good-appearing ears. Outstanding ears should be corrected at about five years of age.

CHIN

Microgenia may be of varying degrees. Fig. 12 illustrates a case of moderate degree of receding chin. Fresh cartilage, preserved cartilage, or ivory may be used for its correction. Some of the most severe types of retrusion of the mandible are associated with ankylosis of the mandibular joint, when the ankylosis occurred in early life. Several pieces of cartilage sewed together and to the mandible may be necessary to produce a good profile, in the more severe types of retrusion.

SCARS

Facial scars should not be repaired for from three to six months following the primary injury. The subcuticular suture is used and has produced good results, Fig. 13.

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A STUDY OF THE COMPARATIVE EFFICIENCY OF BACTERICIDAL AND BACTERIOSTATIC AGENTS IN THE MOUTH

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THE mouth at all times is abundantly supplied with numerous species of bacteria, and practically every known form may be found there (rods, cocci, spiralas, etc.).

The oral cavity represents one of the principal ports of entry for infectious diseases, and the flora does not always remain uniform from hour to hour or day to day. However, less than 1 per cent of the organisms are capable of causing disease. Also many of the organisms found in the mouth may become pathogenic under certain favorable environments and conditions. There is a constant variation of the type and number, and the fluctuation in the potential degree of pathogenicity of the organisms which compose them. The number of organisms is at the maximum at the time of arising in the morning, decreasing after meals, and gradually increasing during the intervals between meals.

There is much evidence to dispute the efficiency of bactericidal agents in the mouth. Studies on the effects of various dentifrices and methods of mouth prophylaxis on the microbial flora of the mouth have shown that sterilization of the oral cavity is practically impossible.

In any plan of mouth prophylaxis, clinical experience teaches that the best results are obtained with those agents that are nonirritating in character. It is also generally known that all of the so-called bactericidal types of agents are irritating to a more or less extent. It is therefore necessary in the treatment of infection in the mouth to depend on a principle other than the mere killing of organisms. It would seem that the environment is of far more importance than the organisms themselves. Many types of organisms are present because of the environment in a particular lesion of the mouth. For instance, in the treating of a pericoronal area of acute infection about a mandibular third molar, our clinical experience shows that the most efficacious type of treatment is to irrigate the wound with a normal saline solution or other similar nonirritating solution and place a wick of cotton or gauze saturated with 5 per cent aqueous solution of mercurochrome in the pocket between the crown of the tooth and the overlying gum tissue. Rodriquez,[21] Simmons,[22] and others have shown that germs grow in some of these mercurochrome solutions and that the mercurochrome is not bactericidal in effect. We do know, however, that it is bacteriostatic, and there is some possibility that its efficiency depends on its nonirritating properties and its ability to cleanse the area of infection mechanically and chemically. On the other hand, in these cases when even a diluted form of iodine is used (a solution known to have a more

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positive bactericidal effect) there are definite irritation and exaggeration of the inflammatory condition with delayed improvement. Treatment of so-called Vincent's infection also impresses one with the vast number of drugs that can be successfully used and lessens one's confidence in the efficiency of any of these drugs when used for oral infections. It appears to be a more physiochemical process of changing the environment by cleansing the area involved. In the case of an osteomyelitis of the bone, operators of experience soon lose confidence in any drug having a specific action upon the organisms when applied locally in the mouth or bone cavity and depend for recovery upon adequate drainage, aeration of the tissues, and building up the patient's general physical condition.

There have been many experimental tests made regarding the efficiency of the so-called antiseptic drugs used in the tooth socket after extraction to kill organisms and promote prompt healing.

Claflin,[4] in a study on the healing of disturbed and undisturbed extraction wounds which were infected artificially and which were treated with a mixture of phenol, glycerine, iodine, and collodion, found no appreciable difference from those treated with the individual drugs, nor could such a difference be observed between treated and untreated infected wounds.

Gardner,[9] in a study of postoperative consideration regarding extraction of teeth, found that "it is better not to attempt to sterilize the field of operation unless a chemical can be used which does not contribute to the cause of inflammation." He found it not advisable to use socket medication in any form since irritation and not sterilization was the result.

It has been the experience of most of these men that the use of any drug in the socket of the tooth retards the healing process. The best results have been attained by those who depend upon clean surgery and thorough removal of areas of infection with a minimum amount of trauma, keeping the saliva out of the wound during the operative process as much as possible, and by depending upon this clean surgery and a clean blood clot rather than antiseptic drugs to take care of the wound. After the removal of the tooth a piece of gauze or cotton is placed over the wound, and the patient is instructed to bite down upon it, holding it firmly in place. This may be repeated until the wound fills with blood and the blood clot is started.

In the study of oral hygiene one of the problems that has attracted the attention of investigators has been the efficiency of dentifrices in mouth prophylaxis. According to Appleton[1] the use of a nonmedicated tooth paste and mouthwash may reduce the number of bacteria by about 40 per cent, but this decrease is of relatively short duration, the count returning to the original within a short time after prophylaxis. The incorporation of drugs or medicaments in dentifrices has been constantly opposed, based on the fact that concentrations effective for bacterial destruction would also destroy oral tissues. Of late, however, antibacterial substances such as hexylresorcinol, sodium ricinoleate, and certain soap substitutes have been quite extensively used in commercial tooth pastes and mouthwashes. Leonard and Feirer[16] examining forty-one different commercial tooth pastes found that none of the dentifrices tested effected any significant reduction in the bacterial content of mouth

washings. In a subsequent publication the same authors reported on the efficacy of a group of alkyl resorcinols in oral antisepsis, and hexylresorcinol (S.T. 37) was found to be the most powerful antiseptic of the group studied, effecting practically complete disinfection of the gum margin in five minutes.

My experience has repeatedly demonstrated that the employment of substances which absolutely destroy microorganisms is contraindicated on account of their deleterious effect upon oral tissue. On the other hand, it was found that less powerful agents failed to exert even a bacteriostatic effect, and hence to establish the desired condition of oral antisepsis.

Among the major obstacles encountered by investigators in their search for the ideal disinfectant was the constant variation in the type and number of the oral flora, and the fluctuation in the potential degree of pathogenicity of the microorganisms which composed it. For instance, it was observed that the number of microorganisms attained its maximum at the time of arising in the morning, decreased after meals, and gradually increased during the intervals between the latter. Moreover, Goadby[11] succeeded in modifying the essential character of the oral flora through the institution of a rigid hygienic and dietary regimen, while Howitt and Fleming[15] noted differences in the oral flora of the same individuals on different days. Also Hine and Bibby[14] found that wide variations occurred from time to time in the bacterial flora of certain portions of the mouth. In the course of a comparison of the bacterial flora of different mouths Bibby[2] revealed variations of such magnitude as to preclude the possibility of the demonstration of a relationship between the nature of the oral flora and the condition of the mouth in which it existed.

Another formidable difficulty consisted in the discovery of a disinfectant agent which would fulfill the fundamental requirements of entire harmlessness to the oral mucous membrane, the teeth, and the body in general; sufficient bacteriostatic or bactericidal potency within the oral cavity and the posterior pharyngeal space; and an agreeable taste and odor. Accordingly, all substances which exhibited a markedly toxic effect were excluded on account of the risk of their absorption by the buccal mucous membrane as well as because of the free communication of the oral cavity with the extensive absorptive areas of the digestive tract. Likewise, caustics or escharotics of even moderately strong concentration were eliminated, since agents which impair the vital energy of somatic cells ultimately facilitate the development of microorganisms.

Results of initial experiments with chemical disinfectants intended for use in the oral cavity were reported in Germany in 1892 by Miller,[18] who tested in turn bichloride of mercury, hydrogen superoxide, iodine trichloride, salicylic acid, permanganate of potash, carbolic acid, benzoic acid, and hydrochloric acid. Of these, bichloride of mercury in a concentration of 1:2,500 proved to be the best disinfectant for the purpose, but like most of the other antiseptics involved in the experiments conducted by Miller its employment as a means of sterilization of the oral cavity appeared impracticable because of its highly toxic properties.

It is the general consensus of opinion based upon an accumulation of experimental evidence that complete disinfection of the oral cavity is impossible,

primarily in consequence of the diverse character and variable number of the bacterial flora in which the normal mouth abounds. Therefore, the attention of investigators has, for some time past, been directed toward the search for agents which would serve either to effect a decrease in the microorganisms present in the oral cavity or else to render them inert and thus to diminish their potential pathogenicity.

The attainment of these two paramount objectives was first sought by the incorporation of drugs or medicaments in dentifrices. However, as in the case of the chemical disinfectants previously tested and discarded by Miller and others, here too it was found that concentrations required for destruction of bacteria also destroyed oral tissues. Thereupon experiments with tooth pastes and mouthwashes containing less drastic irritating substances such as hexylresorcinol, sodium ricinoleate, and sundry substitutes for soap were instituted by a number of independent investigators.

The results of these experiments were far from encouraging. Feirer and Leonard[7] found an average percentage increase in count of mouth organisms of about 300 per cent over the initial one during the period. They also found hexylresorcinol most powerful among a group of alkyl resorcinols for the production of a condition of oral antisepsis, since with its use complete disinfection of the gingival margin was obtained in five minutes. Mead[17] showed that sodium ricinoleate, while devoid of bactericidal qualities, as indicated by its phenol coefficient, when employed as a mouthwash reduced the bacterial count and retained it at a low level for a considerable period, in marked contrast to hexylresorcinol, notwithstanding the fact that the latter exhibits a higher phenol coefficient. Some of the newer dentifrices and mouthwashes contain alkyl sulfates. However, Epstein and his associates[6] warned against the potentially deleterious toxic effects of substitutes for soap utilized as detergents in certain dentifrices. The two substances which they tested experimentally on animals, namely, sodium alkyl sulfate and sodium lauryl sulfo-acetate, proved irritating to both superficial and subcutaneous tissues, and particularly to the esophagus and the stomach. Perrine[20] and his colleagues by their experiments illustrated the toxic action of sodium alkyl sulfate upon the ciliated epithelium of the esophagus.

Hatton, Fosdick, and Calandra[13] in a study of the toxicity and rubefacient action of sulfated higher alcohols found that "the degree of surface irritation of human mucous membranes caused by this preparation is less than that of castile soap in the concentration in which it is generally used."

The role of acid fruits and acid fruit juices as salivary stimulants and the immunity to bacterial invasion, conferred by a supply of saliva adequate in quantity and quality, had long been known, and served as a tentative basis for investigations concerning the efficacy of citrus fruits and citrus fruit juices as possible oral antiseptics. In 1933 Nichols, Hatton, and Dougherty[19] undertook to study the behavior of mucin smears on glass slides treated with acids, alkalies, soaps, and substances which contained vitamin C. Of all these reagents, the latter group of substances, comprising tomato juice, orange juice, lemon juice, pineapple juice, grapefruit juice, and apple juice, proved most effective for the removal of mucin films. It is interesting to note that orange juice was more efficient than lemon juice. Since it is generally agreed that

the mucin in the saliva constitutes one of the most important factors in the problem of the thorough cleaning of the teeth, its removal would appear to be a consideration of cardinal significance in oral prophylaxis.

Following this work of the effectiveness of citrus fruit as a cleansing agent, a study of the local effects of citrus fruits upon the bacterial flora of the mouth and the mouth tissues was undertaken by District of Columbia members of the Florida Citrus research group.*

BACTERIOLOGIC EXAMINATION†

Quantitative determinations of the microorganisms present in the saliva of nine normal individuals were made before and after the use of each of the following materials: 1. Sterile distilled water. 2. Orange juice. 3. Grapefruit juice. 4. Product A (hexylresorcinol tooth paste and mouthwash). 5. Product B (Sodium ricinoleate tooth paste and mouthwash). 6. Product C (Liquid dentifrice containing sodium alkyl sulfate). Plain nutrient agar was utilized for the determination of the total number of saprophytic types, tomato juice agar for aciduric organisms, and blood agar for potential pathogens. Normal diets were maintained during the experimental period. The flow of saliva was stimulated by chewing sterile paraffin, and fifteen to twenty cubic centimeters of saliva from each subject were collected into sterile sputum flasks, just before lunch, after lunch, immediately following prophylaxis, and two hours later. Each subject repeated this procedure three times at intervals of two days for each of the materials included in the tests, and an average of the three counts thus obtained was calculated. The samples of saliva were invariably plated out as soon as they were collected. The teeth were brushed with toothbrushes of medium stiffness in such manner that the bristles remained in contact with the teeth while a slight vibratory or oscillatory movement was applied to the handle. The teeth were brushed with each material for two minutes, followed by a rinse of one minute with the respective mouthwash. When water and citrus fruit juices were used, fresh test material was added to the brush several times during the brushing period. Product C, the commercial liquid dentifrice, was applied to the brush as directed by the manufacturer, while the tooth pastes, Products A and B, were squeezed along the entire length of the row of bristles. Thirty cubic centimeters of each respective mouthwash were used to rinse the mouth after brushing.

In the preliminary tests marked discomfort and irritation of the mucous membrane were experienced with the mouthwash (Product A) which contained a 1:1,000 solution of hexylresorcinol. A dilution of the rinse even to 1:4,000 still caused slight discomfort. The mouthwash containing 1 per cent sodium ricinoleate (Product B) when used in full strength also exerted an irritant effect upon oral tissue. Inasmuch as Product C, which contained 2 per cent sodium alkyl sulfate as an active ingredient, was available only in the form of liquid dentifrice, in order to render tests with this material comparable to tests with other dentifrices a 10 per cent solution of the product was employed as a rinse.

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*Doctors S. F. Acree, M. A. Elliott, Division of Chemistry, Bureau of Standards; L. H. James, J. E. Faber, and H. J. Florestano, Department of Bacteriology, University of Maryland; and Sterling V. Mead and his staff.

†Bacteriologic and chemical data presented in this paper are taken from a report by H. J. Florestano, M. A. Elliott, and J. E. Faber, "The Effect of Citrus Juices and Various Mouth Prophylaxes on the Oral Flora and Saliva," now in press.

The citrus juices used in the experiments were obtained from fruits grown in Florida. The juices for all tests were freshly extracted with a squeezer of the lever type operated by hand, and care was taken to avoid too great a pressure so as to eliminate the oil of the skin as far as possible from the juice.

Immediately following prophylaxis each toothbrush was sterilized by immersion in a solution containing approximately 600 parts per million of available chlorine which had previously been demonstrated as capable of effecting complete sterilization within ten minutes. Each brush was thoroughly rinsed in sterile water before use.

For the purpose of determination of the relative efficiency of the various materials involved in the tests, the average counts obtained immediately after lunch were adopted as a base of 100 per cent, and the decrease following prophylaxis was computed therefrom in terms of percentage on each medium. Similarly the increase in counts two hours after prophylaxis was determined in terms of percentage, with the average of the separate counts immediately following prophylaxis serving likewise as the base of 100 per cent. By the application of this method of calculation it was found that the average percentage decreases in bacterial counts immediately after prophylaxis for Products B and C was 89.2 and 85.7 respectively of the counts obtained after lunch. With Product A the decrease was 59 per cent; with grapefruit juice, 55.8 per cent; and with orange juice and water alike, 46.7 per cent. Accordingly while Products B and C proved most effective for the actual removal of microorganisms, they appeared least effective as inhibitors of bacterial activity, since when counts obtained immediately after prophylaxis were adopted as a base of 100 per cent for the determination of the average percentage increase two hours later, the citrus fruit juices were found to be most efficacious for the inhibition of the action of microorganisms. The increase in the counts was only 169.8 per cent and 205.1 per cent for orange juice and grapefruit juice, respectively. On the contrary, with Product A the increase in the count amounted to 313.4 per cent, with Product C, 331.3 per cent, and Product B, 687.1.

A complete summary of the results obtained from the experiments described above is presented in the accompanying tables and figures. Each column in the figures represents the bacterial count per cubic centimeter of saliva expressed in terms of percentage. The legends attached to the tables and figures are self-explanatory and require no additional comment.

The nine individuals used for these tests were selected because of the almost perfect condition of their teeth and oral tissues. Colored photographs and full mouth roentgenograms were taken both before and after the experiments.

Upon completion of the work and all during the time citrus was used there was not any apparent irritation or change of the oral tissues.

This work is now being followed by further study in mouths showing dental pathosis.

CHEMICAL ANALYSIS

The buffer capacity (or buffer value) was determined by making a hydrogen-ion concentration titration curve with normal acid and alkali on 10 c.c. of saliva and dividing the equivalents of added acid (calculated per liter of saliva) by the change in the negative logarithm of the hydrogen-ion concentration, i.e., change in the pH.

Buffer capacity and hydrogen-ion concentration were determined on all saliva samples used in this work. The hydrogen-ion concentration measurements were made with a commercial glass electrode apparatus, the glass electrode being calibrated with buffer solutions standardized by means of the hydrogen electrode. When measuring the hydrogen-ion concentration of saliva samples, special care was taken to agitate the sample after it was in contact with the glass electrode to be sure that a constant and reproducible reading was always obtained.

Surface tension measurements were also made on part of the saliva samples. These were made at room temperature with a Cenco-duNuoy interfacial tension balance No. 10403. A platinum-iridium ring of 6 cm. mean circumference was used with this tensiometer for the surface tension measurements. It was calibrated using the value 73.0 dynes per cm. for water at 22° C.

A comparison of the hydrogen-ion concentration titration curves for pure citric acid and for orange juice indicates that at least 90 per cent of the acid in the juice is citric.

The hydrogen-ion concentration titration curves on saliva show that its buffer capacity is raised 10 to 20 per cent five minutes after the use of orange juice (instead of water) as a prophylaxis in brushing the teeth, and there is but little change in the hydrogen-ion concentration of the untitrated saliva thus showing that the increase in buffer capacity is due to stimulation and not to the possible accidental contamination of the saliva by orange juice. This increase in buffer capacity is not apparent on samples taken two hours after brushing.

Saliva shows roughly one-fourth to one-third the buffer capacity of the juice from pineapple and oranges. The saliva hydrogen-ion concentration titration curves are fairly straight from hydrogen-ion concentration 3.5 to hydrogen-ion concentration 10.5.

From a comparison of the hydrogen-ion concentration titration curve of a given sample of orange juice with that of pure citric acid, it is possible to estimate the total citrate in the orange juice and the distribution of this citrate between free citric acid and salts of citric acid. Since it is these salts of citric acid that give the citrus juice their alkalizing capacity, it is possible to estimate this alkalizing capacity from the hydrogen-ion concentration titration curve.

SUMMARY OF RESULTS

The effectiveness of the various materials in the actual removal of microorganisms from the mouth will be apparent from the figures in Table I. It will be observed that Products B and C effected the greater percentage decrease in microorganisms; the least percentage removal being obtained with orange juice and distilled water. When the percentage decreases on all culture media were averaged (Table II), it was found that the results with water were the same as with orange juice, while the decrease with grapefruit juice was almost as great as with Product A, which contained hexylresorcinol as an active ingredient.

Although Products B and C were the most effective in actual removal of organisms, they were not so effective in inhibiting further microbial activity during the two hours immediately following prophylaxis (Table III). When the

TABLE I

PERCENTAGE DECREASE IN BACTERIAL COUNTS PER C.C. OF HUMAN SALIVA AFTER VARIOUS MOUTH PROPHYLAXES

PROPHYLAXIS	DECREASE DUE TO PROPHYLAXIS*		
	MEDIUM (AGAR)		
	NUTRIENT (%)	TOMATO JUICE (%)	BLOOD (%)
Water	45.1	53.0	42.1
Orange juice	46.7	50.1	43.3
Grapefruit juice	60.9	48.6	57.9
Product A	50.9	61.1	64.9
Product B	86.7	89.9	91.1
Product C	79.2	87.5	90.3

*Counts after lunch before prophylaxis as 100% base. Product A, hexylresorcinol tooth paste and mouthwash (1/4000). Product B, sodium ricinoleate tooth paste and mouthwash (1 per cent). Product C, liquid dentifrice, active ingredient 2 per cent sodium alkyl sulfate; mouthwash (2 per cent).

TABLE II

PERCENTAGE DECREASE IN BACTERIAL COUNTS PER C.C. OF HUMAN SALIVA AFTER VARIOUS MOUTH PROPHYLAXES

(THE PERCENTAGE DECREASES ON ALL CULTURE MEDIA ARE AVERAGED)

	PROPHYLACTIC AGENT					
	WATER	ORANGE JUICE	GRAPEFRUIT JUICE	PRODUCT A	PRODUCT B	PRODUCT C
Decrease due to prophylaxis (per cent)*	46.7	46.7	55.8	59	89.2	85.7

*Counts after lunch before prophylaxis as 100 per cent base.

TABLE III

PERCENTAGE INCREASE IN BACTERIAL COUNTS PER C.C. OF HUMAN SALIVA TWO HOURS AFTER VARIOUS MOUTH PROPHYLAXES

PROPHYLAXIS	INCREASE TWO HOURS AFTER PROPHYLAXIS*		
	MEDIUM (AGAR)		
	NUTRIENT (%)	TOMATO JUICE (%)	BLOOD (%)
Water	267.3	362.1	176.2
Orange juice	164.3	219.3	125.8
Grapefruit juice	188.9	198.8	227.6
Product A	288.8	262.3	389.2
Product B	635.1	793.4	632.7
Product C	103.5	360.1	530.2

*Counts after prophylaxis as 100 per cent base.

TABLE IV

PERCENTAGE INCREASE IN BACTERIAL COUNTS PER C.C. OF HUMAN SALIVA TWO HOURS AFTER VARIOUS MOUTH PROPHYLAXES

(THE PERCENTAGE INCREASES ON ALL CULTURE MEDIA ARE AVERAGED)

	PROPHYLACTIC AGENT					
	WATER	ORANGE JUICE	GRAPEFRUIT JUICE	PRODUCT A	PRODUCT B	PRODUCT C
Increase two hours after prophylaxis (per cent)*	268.5	169.8	205.1	313.4	687.1	331.3

*Counts after prophylaxis as 100 per cent base.

percentage decreases shown on all culture media for saliva taken just after prophylaxis were averaged and used as a 100 percentage base for determining the average percentage increase two hours later, it was observed that the citrus juices were the most effective in inhibiting bacterial growth (Table IV). Although water brought about as great a decrease after prophylaxis as did orange juice (Table II) it will be seen from Table IV that the percentage increase with water two hours later was 98.7 per cent greater than with orange juice. That citrus juices exert some inhibitory action on bacteria is again shown in a comparison of results obtained with grapefruit juice and Product A. Both materials effected practically the same percentage removal of microorganisms (Table II), yet the percentage increase in counts with Product A two hours after prophylaxis was 108.3 per cent more than with grapefruit juice (Table IV). The marked percentage increase in counts with Products B and C will also be observed in Table IV.

The results of 582 hydrogen-ion concentration determinations and a similar number of buffer capacity values on the saliva of the nine individuals studied are summarized in Tables IX and X. No change in the average hydrogen-ion concentration of saliva was apparent immediately following the use of grapefruit. While a decrease of 0.05 hydrogen-ion concentration was observed when orange juice was employed, this is too small to be of significance. In both cases, the average hydrogen-ion concentration of saliva two hours after prophylaxis was slightly greater than before. Following the use of Products A, B, and C, a comparatively marked increase in the average hydrogen-ion concentration of saliva was observed. The increase with each material was of a greater order than with either of the citrus juices, and remained relatively greater for an interval of two hours after prophylaxis. It will be noticed from Table X that the use of citrus juice gave rise to a temporary increase in the buffer value of the saliva, the increase with orange juice being 0.34 and with grapefruit juice 0.61. Table VIII shows that this increase disappeared within one-half hour after its occurrence. It was thought that the higher buffer value might possibly have been due to the presence of residual citrus juice in the mouth. However, since there was no significant difference in hydrogen-ion concentration of the respective samples taken immediately before and five minutes after prophylaxis, the increase in buffer value was believed to be real.

The results of surface tension measurements of saliva are presented in Table IX. It will be noticed that orange juice effected no great change in surface tension while a marked decrease occurred following the use of Products B and C. The prophylactic agents causing the greater lowering of surface tension of saliva were, in general, the more efficient in removing bacteria from the mouth (Table II).

DISCUSSION

Study of the oral cavity has shown that the mouth is more or less naturally the habitat of a large number and variety of microorganisms varying in counts from hour to hour and day to day.

Sterilization of the oral cavity by means of medicated dentifrices and mouthwashes has not been accomplished. Even when the bacterial count is lowered by artificial aids, laboratory tests have shown that this decrease lasts for only a

short period. Aside from the use of artificial means of prophylaxis the maintenance of oral health is normally assisted by the functioning of the different parts of the mouth and by deglutition. The marked influence of eating on the microbial content of the mouth has been reported by Crowley and Rickert,[5] with which our results have agreed. In some cases we have observed as high as an 88 per cent decrease in number of oral organisms immediately following a meal.

The efficacy of the incorporation of bactericidal substances in tooth pastes and mouthwashes is negligible. Concentrations sufficient to kill bacteria apparently have deleterious effects on the oral tissue. If a minimum concentration of antibacterial agent is used, there is then to be considered the influence of the saliva upon further dilution of the agent. It would seem that even a small inhibition of bacterial growth in the mouth would be a valuable aid in maintaining oral health. This we have apparently been able to do with orange and grapefruit juice, when used both as a dentifrice and mouthwash. The citrus juices

TABLE V

COMPARISON OF EFFECTS OF VARIOUS MOUTH PROPHYLAXES ON BACTERIAL COUNTS PER C.C. OF HUMAN SALIVA EXPRESSED AS PER CENT

AVERAGE PERCENTAGE OF NINE CASES STUDIED*

PERIOD	MEDIUM (AGAR)	MOUTH PROPHYLAXIS						
		NONE (1)	WATER	ORANGE JUICE	GRAPE-FRUIT JUICE	A (2)	B (3)	C (4)
After lunch	Plain nutrient	100	100	100	100	100	100	100
	Tomato juice	100	100	100	100	100	100	100
	Blood	100	100	100	100	100	100	100
After brushing	Plain nutrient	---	54.9	53.3	39.1	49.1	13.3	20.8
	Tomato juice	---	47.0	49.9	51.4	38.9	10.1	12.5
	Blood	---	57.9	56.7	42.1	35.1	8.9	9.7
2 hours after brushing	Plain nutrient	274.5	211.0	145.1	113.9	143.1	35.6	33.7
	Tomato juice	318.9	215.4	146.5	151.8	137.8	30.7	35.9
	Blood	238.8	154.6	116.4	136.6	144.1	23.8	39.8

*Average counts from "after lunch" period used as 100 per cent.

(1) Figures in third block represent counts, expressed in per cent, of saliva samples taken two hours after lunch.

(2) Solution diluted with three parts of distilled water.

(3) Mouthwash contained 1 per cent sodium ricinoleate. Figures represent averages of seven cases studied.

(4) Product exists on market only as a liquid dentifrice. A rinse (1:10 dilution) was prepared directly from the liquid, using distilled water. Figures represent averages of seven cases studied.

TABLE VI

PREDOMINANT TYPES OF ORGANISMS

On nutrient agar:

1. Staphylococci, predominant.
2. Gram + diplococci.
3. Streptococci.

On tomato agar:

1. Long chained streptococci.
2. Aciduric rods (oral L. acidophilus ?).

On blood agar:

1. Alpha—hemolytic type, predominant.
2. Beta—hemolytic type, present in about 1-3 per cent.
3. Indifferent type streptococci.
4. Few staphylococci.

not only proved effective in their inhibitory action, but also, no oral discomfort was experienced by any of the subjects tested. On the other hand, the commercial products not only failed to check microbial activity in the mouth, but two in particular proved quite irritating to the oral mucous membrane.

The efficacy of Products A, B, and C in the removal of microorganisms from the mouth immediately after prophylaxis is apparently due to the presence of a surface tension reducent in each of the materials. The role played by surface tension in oral antisepsis and the mechanisms involved have been reported on by Feirer and Leonard.[7]

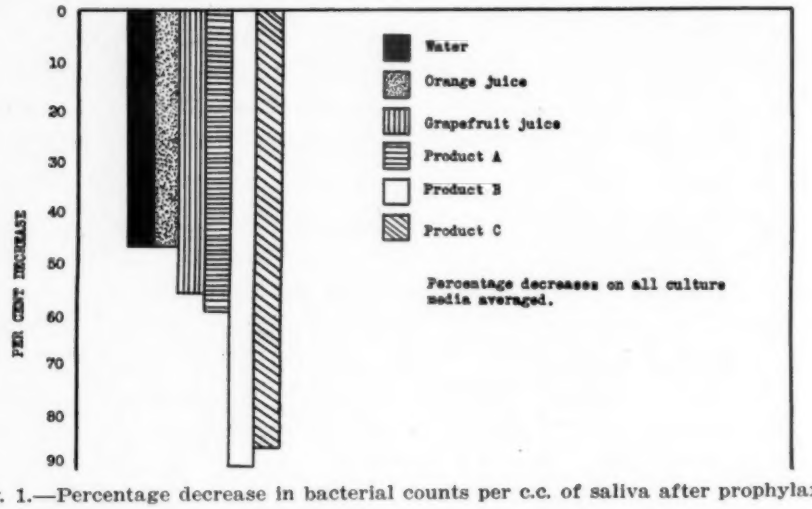

Fig. 1.—Percentage decrease in bacterial counts per c.c. of saliva after prophylaxis.

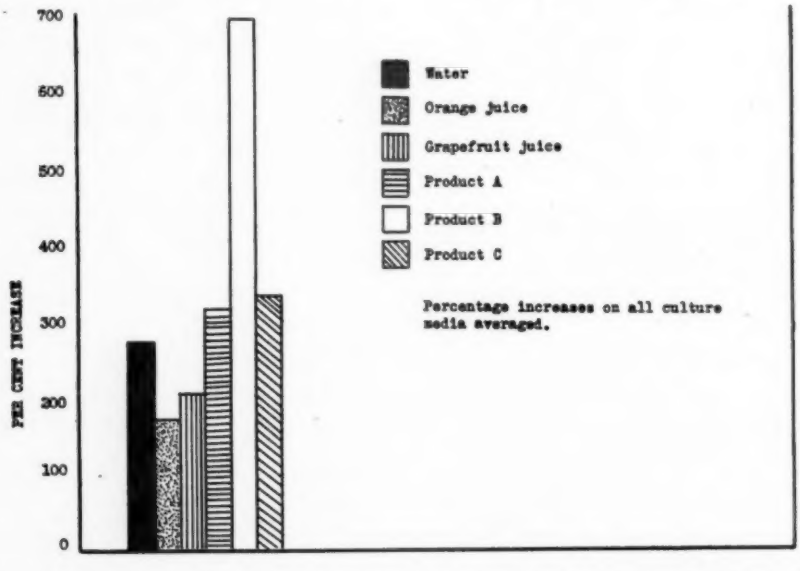

Fig. 2.—Percentage increase in bacterial counts per c.c. of saliva two hours after prophylaxis. phylaxis.

Comparison of results obtained with Products A and B presented an interesting situation. Recalling that Product A contained hexylresorcinol, and Product B sodium ricinoleate, the percentage decrease due to prophylaxis with Product B was more than one and one-half times as great as with Product A

(Tables I and II), despite the fact that hexylresorcinol possesses a higher phenol coefficient. However, in the two-hour interval following prophylaxis (Tables III and IV), Product A maintained a lower percentage increase than did Product B. Carroll[3] and Mead[17] likewise found that sodium ricinoleate effected a greater decrease in the bacterial count of the mouth than did hexylresorcinol. Mead believed that the anti-bacterial action of sodium ricinoleate was due to its unusual cleansing powers and peptizing action.

Gies[10] has stressed the importance of the use of an acid dentifrice for the satisfactory removal of bacterial plaques. He believed that although neutralization of mouth acid should be sought for in prophylaxis, this would necessarily follow by virtue of an increase in flow of alkaline saliva as the result of brushing the teeth with a favorable acid agent. Gies maintained that organic acid, such as is present in fruits like orange and lemon, has this ability to stimulate

TABLE VII

AVERAGE VALUES OF HYDROGEN-ION CONCENTRATION AND BUFFER CAPACITY OF HUMAN SALIVA BEFORE AND AFTER VARIOUS MOUTH PROPHYLAXES

PERIOD	PROPHYLAXIS													
	NONE		WATER		ORANGE JUICE		GRAPE-FRUIT JUICE		PRODUCT A		PRODUCT B		PRODUCT C	
	pH	B. V.*	pH	B. V.	pH	B. V.	pH	B. V.	pH	B. V.	pH	B. V.	pH	B. V.
Before lunch	7.66	8.57	7.42	7.60	7.47	7.51	7.47	7.75	7.42	8.81	7.29	7.72	--	--
After lunch	7.63	8.04	7.62	6.75	7.49	6.86	7.61	7.33	7.49	8.25	7.41	7.43	7.39	7.32
5 minutes after brushing	--	--	7.72	6.03	7.44	7.20	7.61	7.94	7.90	7.89	8.19	10.26	7.92	7.45
2 hours after brushing	7.67	7.72	7.64	6.63	7.65	6.85	7.68	7.18	7.71	7.94	7.74	6.44	7.76	6.77
No. individual samples averaged for each of above recorded values	9		15		27		27		27		27		21	

*Buffer values times 1000.

TABLE VIII

AVERAGE VALUES OF HYDROGEN-ION CONCENTRATION AND BUFFER CAPACITY OF HUMAN SALIVA AT VARIOUS INTERVALS FOLLOWING MOUTH PROPHYLAXES

PROPHYLAXIS	AFTER LUNCH		INTERVAL FOLLOWING PROPHYLAXIS										NO. OF EXPERIMENTS AVERAGED FOR EACH VALUE
			5 MIN.		30 MIN.		1 HR.		1½ HR.		2 HR.		
	pH	B. V.*	pH	B. V.	pH	B. V.	pH	B. V.	pH	B. V.	pH	B. V.	
Orange juice	7.49	6.70	7.23	7.26	7.67	6.64	7.75	6.82	7.73	6.66	7.76	6.70	6
Product B	7.31	7.36	8.84	9.44	7.80	6.79	7.82	6.37	7.80	6.14	7.79	6.28	6

*Buffer value times 1000.

TABLE IX

AVERAGE VALUES OF THE SURFACE TENSION OF SALIVA AT VARIOUS INTERVALS FOLLOWING MOUTH PROPHYLAXES (EXPRESSED IN DYNES/CM.)

PROPHYLAXIS	AFTER LUNCH	INTERVALS FOLLOWING PROPHYLAXIS					PER CENT LOWERING 5 MINUTES AFTER PROPHYLAXIS	NO. OF EXPERIMENTS AVERAGED FOR EACH VALUE
		5 MIN.	30 MIN.	1 HR.	1½ HR.	2 HR.		
Orange juice	55.1	55.2	55.3	56.1	56.5	56.5	0	2
Product B	52.8	45.4	49.2	51.7	52.7	52.8	14	3
Product C	51.6	43.7	--	--	--	48.2	15	10

TABLE X

CHANGE IN AVERAGE VALUES OF HYDROGEN-ION CONCENTRATION AND BUFFER CAPACITY OF HUMAN SALIVA BEFORE AND AFTER VARIOUS MOUTH PROPHYLAXES

(DATA FROM TABLE IX)

PROPHYLACTIC AGENT	pH OF PROPHYLACTIC AGENT	TIME ELAPSED AFTER USE (MIN.)	CHANGE IN pH OF SALIVA	CHANGE IN BUFFER* VALUE OF SALIVA
		5	−0.05	+0.34
Orange juice	3.6	120	+0.16	−0.01
		5	0.00	+0.61
Grapefruit juice	3.2	120	+0.07	−0.15
		5	−0.41	−0.36
A	8.5	120	+0.22	−0.31
		5	+0.78	+2.83
B	8.9	120	+0.33	−0.99
		5	+0.53	+0.13
C	7.3	120	+0.37	−0.55
		5	+0.10	−0.72
Water	7.0	120	+0.02	−0.01

*Buffer values times 1000.

markedly the flow of a highly alkaline saliva. Hanke,[12] in a study of the relation of the buffer value of saliva to dental caries, found that in caries-susceptibles the saliva was usually acid and poorly buffered, whereas the saliva of individuals immune to caries was alkaline and highly buffered. Our results have shown that the local application of citrus fruit juices apparently stimulates a flow of saliva that is highly alkaline and well-buffered. Although the increase in buffer capacity lasted for a short time only, a real change in the saliva was nevertheless produced. It is believed by us that consistent use of citrus products may effect a more permanent change not only in the oral flora but in the saliva as well. Experiments are now being conducted to determine the extent of such changes when citrus fruit is incorporated in the diet. There is a local bacteriostatic action of these juices with apparently no irritation whatever of the tissue, and there is a stimulating effect upon the saliva increasing the buffer value.

It would appear that their use internally will have some local benefit as well. The stimulating and cleansing effect and the local and general stimulating effect of citrus juices between meals is well known. Many of the Latin-American countries utilize fruit in the diet at the end of a meal, which has many factors to recommend it.

There is considerable evidence to show that use of cane sugar and desserts containing sugar is a possible factor in the production of caries and periodontal lesions of the gum. By using citrus fruit it is possible to eliminate some of the harmful effects of sweets as well as aid nature in cleansing the mouth, by their use at the end of the meal instead of producing an unfavorable environment with starchy and sticky desserts.

CONCLUSIONS

1. It is, no doubt, not intended that the healthy mouth should be free from micro-organisms.

2. It would appear that dentifrices and mouthwashes should be used for their physiochemical action, utilizing those types that are free of irritation and injury to the soft tissue, and placing little credence in the bactericidal effects.

3. The mouth environment rather than the mere presence of organisms is of greatest importance.

4. The local application of citrus fruit juices apparently stimulates the flow of a more alkaline and well-buffered saliva.

5. Citrus fruit juices effected no great change in surface tension of saliva, while the most effective commercial dentifrices caused a marked decrease. The prophylactic agents causing the greater lowering of surface tension of saliva were the more efficient in their bactericidal effects in the mouth.

6. It is quite apparent from the evidence adduced in the present study that freshly extracted citrus fruit juices possess considerable value as a dentifrice as well as a mouthwash, chiefly by virtue of their demonstrated cleansing and bacteriostatic effect upon the oral flora and their nonirritant character.

REFERENCES

1. Appleton, J. L. T.: Bacterial Infection, Philadelphia, 1923, Lea and Febiger.
2. Bibby, B. G.: A Comparison of the Bacterial Flora of Different Mouths, J. Dent. Research **17**: 423, 1938.
3. Carroll, B. S.: A Study of Dentrifices, Unpublished data, 1933.
4. Claflin, Roland S.: Healing of Disturbed and Undisturbed Extraction Wounds, J. A. D. A. **23**: 945, 1936.
5. Crowley, M. C., and Rickart, U. G.: A Method for Estimating the Bacterial Content of the Mouth by Direct Count, J. Bact. **30**: 395, 1935.
6. Epstein, Sidney, Throndson, A. H., Dock, William, and Tainter, M. L.: Possible Deleterious Effects of Using Soap Substitutes in Dentrifices, J. A. D. A. **26**: 1461, 1939.
7. Feirer, W. A., and Leonard, V.: Hexylresorcinol in Oral Antisepsis With Special Reference to Solution S. T. 37, Dent. Cosmos September, 1927.
8. Feirer, W. A., and Leonard, V.: Oral Hygiene. I. The Diurnal Tide of the Bacteria of the Mouth and Its Use in Determining the Actual Value of Preparations Commonly Employed as Oral Antiseptics, Dent. Cosmos, April, 1931.
9. Gardner, B. S.: Postoperative Considerations Regarding Extraction of Teeth, J. A. D. A. **16**: 235, 1929.
10. Gies, W. J.: The Dentrifrice Problem, J. Nat. D. A. **8**: 851, 1921.
11. Goadby, K. W.: Diseases of the Gums and Oral Mucous Membrane, Brit. M. J. **2**: 769, 1910.
12. Hanke, Milton J.: Diet and Dental Health, 1933, Chicago, The University of Chicago Press.
13. Hatton, E. H., Fosdick, L. S., and Calandra, J.: The Toxicity and Rubefacient Action of Sulphated Higher Alcohols, J. Dent. Research **19**: 87, 1940.
14. Hine, M. K., and Bibby, B. G.: Variations in the Oral Flora From Time to Time, J. Dent. Research **18**: 61, 1939.
15. Howitt, B., and Fleming, W.: The Quantitative Examination of the Mouth Flora Under Different Dietary Conditions, J. Dent. Research **10**: 33, 1930.
16. Leonard, V., and Feirer, W. A.: Oral Hygiene, An Examination of the Alleged Antiseptic Properties of Toothpaste, Dent. Cosmos, June, 1927.
17. Mead, S. V.: A Study of The Bactericidal, Bacteriostatic and Peptizing Action of Certain Dentifrices, J. Dent. Research **16**: 41, 1937.
18. Miller: Die Mikroorganismen der Merndohle, Leipsig, 1882.
19. Nichols, Anna C., Hatton, Edward H., and Doherty, J. A.: A Study of the Behavior of Mucin Smears Treated by Acids, Alkalis, Soaps and Substances Containing Vitamin C, J. A. D. A. **20**: 707, 1933.
20. Perrine, R. L., Throndson, A. H., and Tainter, M. L.: J. Dent. Research **18**: 81, 1939.
21. Rodriquez, F. E.: Mercurochrome and Iodine as Disinfectants of Mucous Membrane of Mouth; Preliminary Report on Relative Effectiveness, J. A. M. A. **91**: 708, 1928.
22. Simmons, J. S.: Bacterial Action of Mercurochrome-220 Soluble and Iodine Solutions in Skin Disinfection; Preliminary Report, J. A. M. A. **91**: 704, 1928.

CONTROL OF HEMORRHAGE

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Etiology

THE causes of hemorrhage are trauma, surgical operations, irritation by foreign bodies and loose bone, sepsis, periodontal disease, invasion of malignant growths, as well as certain constitutional disorders. The types of hemorrhage which are likely to prove most troublesome after operation occur in hemophiliacs or congenital bleeders, patients who have lowered resistance from infection and diseases of the blood, and persons having high blood pressure. According to the vessel involved, hemorrhage may be arterial, venous, or capillary.

Varieties of Hemorrhage

I. According to time.—

(a) A primary hemorrhage is one which occurs at the time of the injury.

(b) An intermediate or recurrent hemorrhage is one which occurs within twenty-four hours after the cessation of the primary hemorrhage.

(c) A secondary hemorrhage is one which occurs after twenty-four hours. It is invariably the result of septic infection which opens the vessels by ulceration or by breaking down the clot. Absence of fibrin and fibrous tissue formation due either to local or constitutional conditions result in secondary hemorrhage.

II. According to cause.—

(a) Traumatic hemorrhage is one which occurs as a result of wounds or injuries to vessels.

(b) Nontraumatic or spontaneous hemorrhage is one which occurs as a result of disease. The vessel walls may be ulcerated or inflamed. There may be a change in the composition of the blood or the elements of the blood. Cook[6] in his article "Blood Dyscrasia from a General Point of View" gives an excellent description of many of these diseases.

In polycythemia (erythemia or Vaquez's disease) the etiology is not known but the onset is usually insidious and chronic. The symptoms are dizziness, headache, and constipation. There is usually a black, red color of the face, mucous membranes, and hands, especially in cold weather. The viscosity of the blood is usually increased. The red cell count is extremely high, from 6 to 10 million with an increase of hemoglobin percentage. There is a tendency to bleed, especially the gums, which is often quite difficult to control.

Hemorrhagic infiltration of the peritonsillar tissues and stomatitis in the form of acute gingivitis and sepsis often occur in agranulocytosis. In pernicious anemia a very fluid and lightly colored drop of blood is obtained upon puncture.

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The viscosity is lowered and the coagulation time is prolonged. In aplastic or agregeneratory anemia, purpura and mucous membrane hemorrhage appear. The red cell count becomes very low and the blood platelets are almost absent. In lymphatic and myelogenous leucemia there is often free bleeding from the gingiva. There may be ecchymosis, petechia, or hypertrophy of the gums. The oral tissues as well as the skin often have a very pale, sallow appearance. Thrombocytopenia (purpura hemorrhagica) is characterized by profuse bleeding especially from the gingival tissue, ecchymosis, and petechia of the mucous membrane of the mouth, prolongation of the bleeding time and normal coagulation rate of the blood. There is a very marked reduction of blood platelets, less than fifteen thousand. There is usually a slight leucocytosis.

Secondary anemias often follow severe hemorrhage. Increase in the blood pressure is often the cause of postoperative hemorrhage.

There are many forms of stomatitis showing bleeding from the gingiva and gums, ranging from a simple gingivitis and other forms of local gingivitis to the various types of systemic disturbances, such as leucemia, etc., among these previously described. Before surgery it is very imperative to make a careful preoperative examination. With many of the types of stomatitis or where bleeding of the gums and gingiva occurs, there is much danger associated with surgery, especially in leucemia, thrombocytopenia, polycythemia, etc.

The operator should be careful and observe his patient well before operating, noting all details about his patient and making good use of his findings before he meets with alarming symptoms. The important fact to bear in mind is that just as the amount of blood in an organism is reduced, so the coagulability of the remaining liquid is lessened; therefore, an anemic person should be treated with care. If the teeth are the cause of infection, of course the septic focus must be removed in all cases, but precautions should be taken to avoid any untoward effects.

Hemophilia.—Hemophilia is a condition of the blood in which a disturbance interferes with coagulation. Only males have the disease, and it is transmitted through the unaffected female. It is a congenital and hereditary disease. The disease runs in families usually, but sporadic cases may occur. The careful operator should always bear in mind the possibility of his patient being a hemophiliac and question him accordingly. If the history of the patient brings out that he suffers from such disturbance, preoperative measures should be considered. Tension sutures are most useful in stopping hemorrhage in these cases.

Cases of this type are distinctly hospital cases, as they need constant watching and treatment which cannot be given at home.

Birch,[3] in a short series of experiments, concludes that urine of males with severe hemophilia is deficient in the female sex hormone, which suggests the value of ovarian therapy.

There is usually a normal bleeding time. Hemophiliacs, or "bleeders," as a rule show no other abnormality except this tendency to hemorrhage; blood platelets appear to be normal in all respects, and there is no characteristic change shown in the red and white cells. The chief symptom of hemophilia is the tendency to bleed, following a minor injury which, in a normal individual, would cause little or no hemorrhage. The vein or ear may be punctured to secure

blood for examination without injury, as hemophiliacs do not bleed from pin-pricks. Prolonged and obstinate epistaxis is a common manifestation of the disease. Serious hemorrhage may follow the extraction of a tooth, and death has followed such a procedure. An effort should always be made to secure a careful family history, which frequently throws light upon this condition. Patients of this type are of a nervous temperament. The clotting time is usually from eight to fifteen minutes longer, and there is a history of extreme bleeding from infancy.

The treatment is the same as for other types of hemorrhage, but preventive and preoperative treatments are the most important.

When an extraction of a tooth for a known hemophiliac is contemplated, it is well first to type his blood, to hospitalize him, to remove the tooth, and to pack the socket with gauze saturated with compound tannic acid solution and suture the edges of the wound. The gauze should not be changed for forty-eight hours, and then it should be gently removed. If there is any tendency to bleed, only a portion of the gauze is at first removed and the remainder removed the following day. The wound should be repacked with gauze and compound tannic acid solution.

If there is any tendency to hemorrhage or continued bleeding, a transfusion of blood is advisable. In some cases, with a bad history, it is well to do the transfusion before the operative procedure.

While females are not true hemophiliacs, it is well to keep in mind that severe hemorrhage may occur or that persistent, capillary oozing may take place in females at times very closely resembling the nature of the bleeding of hemophiliacs.

It is well in all cases to take as much precaution as possible.

III. According to the source of the vessel injured.—

(a) Arterial hemorrhage is bleeding from an artery. The blood escapes in spurts and is of a bright red color. Pressure above the wound, between it and the heart, controls it.

(b) Venous hemorrhage is bleeding from a vein. The blood is dark in color and it flows steadily. Pressure on the vein below the wound checks the bleeding.

(c) Capillary hemorrhage is one which is characterized by a general oozing of blood from the surface.

IV. According to location.—

(a) An external hemorrhage is one in which the blood escapes from the skin or soft underlying parts.

(b) An internal hemorrhage (concealed) is one in which the blood escapes into a body cavity, a hollow viscus, or into the tissues. In such cases no blood may be actually lost from the body.

V. According to severity and danger.—

(a) A severe hemorrhage is one which occurs as the result of the rupture of a large vessel and is accompanied by shock.

(b) A slight hemorrhage is one in which there is only a small amount of blood lost.

(c) A profuse hemorrhage is one which occurs from a very large vessel so rapidly that it is difficult to find the source and control it. A sudden loss of blood is more dangerous than a gradual loss, since the blood vessels have not time to be readjusted.

Factors Which Influence the Amount of Hemorrhage

A patient with high blood pressure usually bleeds more than one in whom the blood pressure is normal. It is important to note the relationship existing between hemorrhage and shock. During shock the blood pressure is low and a hemorrhage may cease, but it may reappear when the patient recovers from shock due to a rise in blood pressure. The amount of hemorrhage is also determined by the nature of the wound and the nature of the blood. Hemophilia, anemia, and jaundice favor hemorrhage.

SYMPTOMS

Bleeding may occur as a slow seeping or as a profuse flowing. The systemic reaction depends upon the severity of the hemorrhage. Loss of blood may cause faintness and nausea. The pulse becomes increasingly more rapid and weak and there is a continued fall of blood pressure. The temperature is usually subnormal unless there is infection. In the later stages the respiration becomes rapid and feeble, the skin and mucous membrane become pale and cold, and dizziness and headache occur. The loss of blood may result in death if it is not checked.

Diagnosis.—The cause and location of hemorrhage should be immediately ascertained as the treatment will vary according to the cause.

PREOPERATIVE CONTROL OF HEMORRHAGE

Preoperative control of hemorrhage demands that in cases in which there is an abnormal tendency to bleed, an attempt be made to restore the condition of the patient to normal before the operation. The ideal method is to have a physical examination including a blood examination with the hemoglobin content and red blood cell count, leucocyte count, differential, clotting time, and bleeding time. There are prophylactic measures. While it is true that sometimes with a bleeding or coagulation time of three minutes hemorrhage will occur, and that in other instances with a bleeding or coagulation time of ten minutes hemorrhage will not occur, still it is possible to find some of the extreme cases by a blood examination and prevent unwittingly getting into unfavorable conditions. A high blood pressure also has a tendency to cause bleeding and this must be given the necessary attention. Where the blood picture denotes that there may be some general disturbance, such as hemophilia or leucemia, these conditions should receive the proper systemic treatment; and where the clotting or bleeding time of the blood is high and no general cause can be ascertained, it is a good practice to use preliminary medication in order to reduce the bleeding and clotting time. Calcium lactate is considered valuable by some, but must be used in large doses. If it is used at all, I would suggest 20 grains three times a day for a period of three days, operating on the fourth day. Calcium gluconate is recommended by some operators. It is pre-

ferred by some to the chloride and lactate because it is less irritating and more palatable. Dosage: Orally for adults, 5 Gm. (75 gr.), three times a day; for children, 2 Gm. (30 gr.), three times a day before meals, in water, orange juice, or tea. When possible the general systemic condition should be improved with the necessary corrective diet with the addition of large amounts of citrus fruits in order to lower the bleeding and clotting time. Large amounts of gelatin are recommended by some.

Brown states that adrenalin 1:1000 given internally in 10 to 30 minim doses, every two or three hours also has beneficial effect and that more prompt results may be secured by the hypodermic injection of 10 to 20 minims.

Coagulin Ciba, intramuscularly or subcutaneously, may be used by injecting 1½ c.c. every other day for three injections.

Thromboplastin and hemostatic serum are recommended by some for local application and also for hypodermic injection.

Intravenous injection of Congo red has been suggested by Graves and Kickham.[12] It is being used rather extensively to prevent prostatic hemorrhage. Shortening of the time required for blood clotting by feeding of vitamin D or ergosterol irradiated with ultraviolet light has been reported by Corson, Erwin, and Phillips as the result of a test made on white rats.

Tainter, Throndson, and Richardson in studies[26, 27] found, "The results of this study, taken with those previously reported in the Journal and from this institution, leave no doubt that the agents commonly advocated for oral administration as hemostatics in dentistry are not effective when so used. The dental practitioner is advised to direct his efforts at controlling hemorrhage to local application of demonstrably effective agents rather than to try to speed up the coagulation process by systemic medication."

As a preoperative measure I would recommend attention to diet, including 30 ounces or more of citrus, improving the general physical condition of the patient, prescribing for any general disorders, rest, and sunshine.

Koagamin is valuable for intramuscular or intravenous injection.

Transfusion is always to be considered in extreme cases.

Locally in cases of capillary bleeding the compound tannic acid solution, coagulation with diathermy, or sutures are of the most value.

Many advocate a blood transfusion as a preoperative measure rather than drugs, and in some cases this is advisable. Because of the danger of anaphylaxis, horse serum should not be used as a preoperative measure.

Study of Blood Elements in Prevention of Hemorrhage

The factors to be considered in a study of prevention of hemorrhage, in the relative order of their importance, are clotting time, bleeding time, prothrombin time, the number of blood platelets, and calcium determination.

Clotting time and bleeding time are more useful than the others. The determination of blood calcium is of questionable value. Coagulation time and prothrombin time tend to agree. The blood platelet count is useful in the diagnosis of purpura.

Before operation, it is best to endeavor to determine the possibility of hemorrhage. Normal blood clots in three to eight minutes. The clotting time

of the blood, however, is not conclusive evidence that there will or will not be hemorrhage. The clotting time and the bleeding time may not be the same. The only real service I can see in the clotting or coagulation time is to ascertain when the coagulation time is extremely high (from ten to fifteen minutes) and where there is more likelihood of hemorrhage.

Method of Determining Bleeding and Clotting Time

A very simple, practical way of determining the coagulation time of the blood is to put several drops of blood from the ear or tip of the finger on a clean glass slide. The blood should flow from the part without squeezing, as squeezing lessens the clotting time, due to a mixture of blood with tissue fluid. The point of a clean needle is drawn through one or another of the drops at one-minute intervals until there is a distinct fiber which will adhere to the needle a little before the true clot is formed. Another method which I prefer is to use a capillary tube, one and one-half millimeters on its outside diameter. After nicking the tip of the ear or the finger, the blood is allowed to run into the capillary tube. A short section of the tube is broken off at one-minute intervals. As soon as coagulation occurs, the fibrin is seen stretching between the broken ends of the tube. The tube should be gently scratched with a file before breaking. The bleeding time is ascertained by timing and observing the termination of bleeding at the point of puncture. Various other methods may be used, such as Howell's method, and Lee's and White's method. These methods, however, require blood from a vein.

Citrus Fruit

The literature is replete with reports showing that citrus fruit has a definite effect in the treatment of hemorrhagic condition which has been attributed to vitamin C in this fruit. It is well known that in scurvy there is a definite clinical condition showing hypertrophy, inflammation, congestion, and bleeding at the gingival margin with dark, bluish-red discoloration of the tissue. There are cases of scurvy where the gingival tissues cover more than half of the crown of the tooth. On the other hand, there are many cases of subclinical scurvy which are not sufficiently marked in their clinical appearance to be easily recognized by the patient, physician, or the dentist. It is only when these conditions become exaggerated that they are usually recognized as a diet deficiency. In the case of scurvy the treatment known for years has been the administration of lemon juice, orange juice, grapefruit juice, tomato juice, and cabbage juice, etc. Accompanying these cases of scurvy there is a hemorrhagic tendency; when these conditions have responded to the administration of increased amounts of citrus fruit, vitamin C is presumed to have accomplished this result. It is significant, however, in the work upon caries and periodontal disease that citrus fruit rather than synthetic vitamin C has been used.

Szent-Gyorgyi[25] and his associates in 1936 reported the presence in extracts of Hungarian red pepper and later in lemon juice of a substance other than ascorbic acid which would control hemorrhage that occurred in certain clinical conditions. These cases consisted of purpura (thrombocytopenia purpura) diabetes, infectious diseases, etc. As a consequence of that work, Szent-

Gyorgyi and his associates demonstrated a substance which restored the capillaries to their normal state. They called this substance vitamin P ("Permeability vitamin"). The active substance was identified as hesperidin. Scarborough and Stewart,[22] in a study of the factors producing petechial hemorrhage, found that the oral administration of hesperidin would reduce the hemorrhage in patients with vitamin deficiency which was without the presence of ascorbic acid in the diet. Moll[18] and other writers disagree with Szent-Gyorgyi[25] in regard to the presence of a vitamin P. Leetch,[15] while agreeing to the importance of vitamin P therapy, suggests that instead of hesperidin the preparation might contain another substance in the flavon group. Lojas[13] claims vitamin P to be of great clinical prophylactic value. Vitamin P, which is also known as citrin (citris flavon) was obtained in the form of crystals, two grams of pure crystal from 70 liters of lemon juice. Zacho[28] used citrin from rose hops for the treatment of hemorrhagic conditions.

Szent-Gyorgyi[25] states, "In the course of various experiments it was possible to observe that the cause of experimental scurvy was not only a deficiency of vitamin C, but rather a combination of avitaminoses due to lack of both C and P. The reason for the failure to discover vitamin P previously is that the particular avitaminosis caused by its lack reveals no physical sign. Meanwhile it has been noted that if the deficiency of vitamin P is accompanied by a deficiency of vitamin C, the signs of the avitaminosis due to lack of vitamin C are essentially modified. It is possible to obtain a definite condition of avitaminosis due to lack of vitamin C if animals are given abundant amounts of vitamin P, and at the same time are deprived of vitamin C. The scurvy thus produced differs materially from ordinary scurvy. The signs of this avitaminosis due solely to lack of vitamin C are recession of the gums from around the teeth and swelling joints, with death toward the sixth to the eighth week. Accordingly, it may be concluded that in ordinary scurvy hemorrhage of the different organs is caused by deficiency of vitamin P, and that rapid emaciation and subsequent death at about the end of the fourth week are the consequences of avitaminoses due to the combined lack of vitamins C and P. Clinical observations suggest that this new vitamin P is of great importance."

In this connection it would appear that there are a number of possibilities for the use of citrus fruit as a preoperative measure for the reduction of bleeding and clotting time. However, this does not yet appear to be a method that can be used for immediate results, as it takes considerable time. In my experimental work in connection with the Bureau of Standards and the University of Maryland for the Florida Citrus Commission,* we have found that it takes months in finding experiments to reach the peak of saturation for vitamin C in the bodies of those greatly deficient in this vitamin. Consequently it is reasonable to believe that it takes considerable time to reach the peak of the vitamin P curve. I have studied a number of cases for this effect and in a few striking cases have obtained a very favorable result. In one case in particular of a young man who has been a patient for a few years, there was always trouble

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with bleeding after removal of teeth. In his case I have never been able to consistently lower the bleeding and clotting time and prevent hemorrhage. It has been necessary to control the bleeding by packing and suture. When he presented himself for another extraction recently, an attempt was made to lower the bleeding and clotting time with calcium lactate and other methods without success. He was then given intensive amounts of grapefruit juice three times a day. In ten days' time, his bleeding and clotting time was reduced to six minutes and the tooth was removed. The wounds healed without hemorrhage and without packing or suturing.

POSTOPERATIVE TREATMENT OF HEMORRHAGE

Local treatment of hemorrhage includes (1) ligation, (2) pressure, (3) sutures, (4) cautery, (5) astringents or styptics, (6) torsion, (7) Position, (8) extreme heat or cold.

Ligation is the method of election when the vessel is large enough. The intima of the vessel is compressed by the ligature and is destroyed. Consequently, at this point the blood coagulates, the coagulum occluding the vessel. The vessel is caught by a forceps and tied with a ligature of catgut. The ligature should be small and should include no more extraneous tissue than is necessary. The selection of a particular type of gut is important, depending on the particular vessel to which the ligature may break through the vessel. Ligation of such a large vessel results in a rise in blood pressure which falls again with the establishment of a collateral circulation.

The permanent ligation of the common carotid artery is an operation that should never be done without necessity, as it is not free from danger.

In surgical operations the loss of blood should be prevented by using hemostats to clamp bleeding areas. Wherever possible, large vessels in the field of operation should be isolated and ligated before being cut.

Pressure may be applied with the fingers (digital pressure), a tourniquet, tight bandage, pad, or compress. Hemorrhage from a large vessel can be controlled temporarily by digital pressure. Aseptic gauze packing held by a tight bandage quickly checks capillary hemorrhage and usually controls hemorrhage from arterioles and venules. Bleeding from bone can be controlled by filling the opening with pieces of crushed muscle, antiseptic wax, or filaments of gauze. Hemostatic forceps are frequently used to crush the ends of small vessels. Often they require no further attention when the forceps are removed.

Suturing is often used to bring together the edges of a wound and thus act as a hemostat. At times one experiences difficulty in ligating the bleeding points, in which case suturing is permissible. In other cases its use is objectionable, for the edges of the wound must be brought so close together in the attempt to occlude the vessels that as a result strangulation of the tissue occurs.

Cautery should not be employed too frequently. It should be applied at a dull red heat and not a white heat for then its action is the same as though one were to use a knife, and hemorrhage results. The cautery is more effective in limiting infection and should be used with this purpose in mind. Cautery by the diathermy method is very valuable in some cases. This method is of great value in gingival hemorrhage due to purpura and leucemia.

Astringents and styptics are seldom used except in cases of emergency, for they produce a tough coagulum which interferes with healing. Adrenalin is used both locally and hypodermically to check bleeding vessels. It is applied to bleeding surfaces on gauze or as a spray in a dilution of 1:1000. Tannic acid is also used. Such astringents as silver nitrate, acetic acid, tannic acid, and ferric chloride precipitate the proteins and thus check bleeding. Koagamin is very efficient.

Torsion is arresting hemorrhage by twisting the cut ends of a vessel. This action causes rupture of the intima and media of the vessel.

Elevation is one of the simplest methods of arresting hemorrhage. In cerebral hemorrhage or in epistaxis the head of the bed is elevated.

Hot and cold applications are often used to check capillary and venous hemorrhage. Heat in the form of very hot water stimulates the muscle fibers of the vessels to contract and coagulates the albumin of the tissues and the blood. Cold in the form of ice or cold water will often check capillary hemorrhage and prevent discoloration, as in contusions.

Treatment of Bleeding from Bone.—In most cases bleeding from bone may be controlled by pressure or packing with gauze saturated in tannic acid solution. Sometimes it is necessary to crush the bone in the bleeding area by tapping a blunt instrument with the mallet. The use of bone paste is also helpful.

I would warn against the use of Monsel's solution, as the clot formed comes away easily and bleeding is started again.

A patient suffering from severe bleeding, after having the socket packed —and this is always the first consideration—should be placed at perfect rest, in an upright position. Cold applications to the jaw have a tendency to constrict the blood vessels.

Bleeding Vessels in Soft Tissue.—Hemorrhage from vessels in the soft tissue may be controlled by digital pressure, packs of gauze saturated with glycerite or tannin, and ligation of the bleeding ends of vessels and tissues en masse by the use of deep sutures. The application of cold packs is useful.

Hemorrhage from Tooth Socket and Bone Cavities.—After the removal of teeth and pathologic conditions associated therewith, the patient should be instructed to bite down upon gauze or cotton until clotting occurs. Mouth washes should not be used for twenty-four hours, as they tend to promote bleeding. Hemorrhage in bone cavities may be controlled by pressing a piece of soft wax firmly into the bone or by the use of adrenalin chloride or glycerite of tannin packs. Slight capillary hemorrhage often may be controlled by putting glycerite of tannin on a piece of gauze and having the patient bite down and keep firm pressure for from ten to fifteen minutes. Hemorrhage from the tooth socket may be controlled by packing with a strip of ½-inch gauze dipped into one of the tannic acid preparations.

The hemorrhage following tooth extraction usually ceases spontaneously. If continued unduly, it must be checked. Excessive hemorrhage following tooth extraction is caused by either local conditions or systemic conditions. The local conditions are as follows: an unusually large alveolar vessel, forcing the alveolar plates asunder while extracting and thus having a gaping wound; a frac-

tured root in the socket acting as a wedge and keeping the alveolar plates apart; injury of a larger vessel, such as the inferior alveolar artery; incomplete removal of pathologic tissue.

In persistent oozing or free bleeding, novocain may be injected or the patient may be anesthetized with nitrous oxide and oxygen and the cavity packed with one-half inch strip of gauze saturated with glycerite of tannin or compound solution of tannic acid; or a few drops of eugenol, dentalone, or essential oil placed on the gauze and then dipped into tannic acid powder. This packing is usually left forty-eight hours and then changed daily until granulation occurs. In patients with high blood pressure or in hemophiliacs, it is often necessary to suture the gum over the packing with catgut or a silk suture. In postoperative hemorrhage the socket or cavity should first be washed out thoroughly with hydrogen peroxide, warm saline solution, or aromatic chlorozene solution, as there is usually a broken-down clot or infection present. Any loose particles of bone or tooth or any other irritation should be removed. Cold packs to the side of the face and small pieces of ice held in the mouth, as in tonsil cases, are helpful.

In some cases of hemorrhage, especially the type of hemorrhage that occurs after tooth extraction, or where there is not a deep wound, the bleeding can be controlled by putting a suture with a slight bit of tension. These catgut sutures with slight tension are also very valuable in bleeding due to hemophilia.

I have found another method to be used in some types of hemorrhage. Where a patient has persistent bleeding around a tooth which may be due to a systemic disturbance, such as leucemia or purpura, and in some of the cases of persistent bleeding from local causes that occur about the gingiva where it is difficult to pack or suture, the monopolar electrothermic current may be used and the tissue coagulated, and thus control the bleeding. This may be done by placing the metal electrode directly upon the point where the coagulation is desired, or the operator may place a hemostat or other instrument upon the bleeding area and touch this instrument with the diathermic electrode. When bleeding is obstinate it is occasionally advantageous to give one-quarter grain of morphine to quiet the patient and prevent pain. This also reduces the anxiety of the patient, lowers pressure of the blood, and makes him more agreeable to treatment required.

Delayed hemorrhage often occurs in the night and may often be controlled by the use of ice bag, by the patient biting on a piece of gauze dipped in strong tea to get the effect of tannic acid in the tea. The patient may dip the tea ball into warm water, place over the wound and close the teeth upon it. This can be changed just as often as necessary to control the bleeding. The patient is put in a semireclining position with an ice bag. This will stop most hemorrhage. Occasionally it is necessary to see the patient.

Compound Solution of Tannic Acid.—Fantus and Dyniewicz[10] concluded that 10 per cent solution of tannic acid is likely to be the most desirable strength and state:

"In view of the fact that the solution is intended to be applied to a raw surface, it seems logical that isotonicity might be desirable, or better, that the ions naturally existing in the tissue juices be present in the solution (isotonia).

This suggests the use of physiologic solution of sodium chloride or, better still, of Ringer's solution as the solvent. We therefore believe that Ringer's solution is the best solvent for the tannic acid.

"While a 10 per cent solution of tannic acid has better keeping qualities than a more diluted solution, after two weeks tannic acid deteriorates rapidly. The conclusion is that benzoic acid as well as salicylic acid is successful as a preservative for the tannic acid solution.

"On the basis of these experiments we therefore recommend the following formula:

Compound Solution of Tannic Acid

Potassium chloride		0.42 Gm.
Calcium chloride		0.84 Gm.
Salicylic acid		1.00 Gm.
Sodium chloride		10.50 Gm.
Tannic acid		100.00 Gm.
Distilled water	to make	1000.00 cc.

Mix and permit to stand with occasional agitation until dissolved and filter, if required to dispense a clear solution.

"This solution is sufficiently stable to be kept on hand for use in the emergencies occasioned by extensive burns.

"We also recommend that 10 per cent silver nitrate solution be applied to the tannic acid crust."

In summary they state: "A solution has been devised and its formula published that has advantages over the tannic acid solution in water for the following reasons:

"1. It is endowed with good keeping qualities.

"2. It produces a denser coagulum.

"3. It has bacteriostatic action, which is absent in the plain tannic acid solution."

I am using a normal saline solution with 10 per cent tannic acid.

Prevention.—Every dentist should have a comprehensive idea of the causes of hemorrhage. The way to prevent hemorrhage is to be exacting in the diagnosis and preoperative preparation. It is much better to cooperate with a physician in regard to the physical condition of the patient, than to start the operation and have to call the surgeon out in the middle of the night to stop a hemorrhage that could often more easily have been attended to by the dentist. The general factors which tend to produce hemorrhage are: (1) hypertension, (2) blood dyscrasias, (3) acute infection, (4) lack of one of the blood elements essential to clotting, (5) low blood calcium, (6) excitement, (7) lacerated wounds, (8) cut or torn vessels. Immediate hemorrhage should be controlled before the patient is allowed to leave the office. This is usually done by having the patient rest. When it is necessary the soft tissues may be sutured over the wound or the wound may be packed. It may be necessary to tie any vessels involved.

SYSTEMIC TREATMENT OF HEMORRHAGE

The constitutional treatment is much the same as for shock and should be given simultaneously with the local treatment. The patient should be put to bed and kept quiet. When the bleeding has been effectively stopped, measures should

be taken to increase the volume of blood. They include direct transfusion of blood, hypodermoclysis, and intravenous infusion of physiologic saline solution. Stimulants should not be given until bleeding has been effectively stopped.

Koagamin.—Steinberg and Brown[24] in their experiments found that extracts from certain plants accelerated the rate of coagulation of the blood. The basic sources of this material were Shepherd's purse, wood sorrel, oxalis, citrous fruit, rhubarb, and euphorbia. From these preparations, Steinberg and Brown isolated monoclinic crystal which were identified as oxalic acid and then commercial oxalic acid was found to possess the same ability to reduce the bleeding time in the rabbit.

Schumann[23] states that there is some reason to believe that there is some correlation between the work of Dam,[7] Schonheyder,[22] Almquist,[2] and others on the so-called vitamin K with the work of Steinberg and Brown. Oxalic acid is found to be a very potent agent in the mechanism of clotting. The extract of certain plant substances containing as its principal agent oxalic acid has been put on the market under the name of Koagamin, which is a sterile solution for intravenous and intramuscular administration. Milliken[17] reports the use of Koagamin in eleven cases and states: "In all the cases after the first one, which has been reported in detail, the coagulation time was taken both before and after injection of Koagamin, and it was found that this time was reduced by one-half. The dose given in most of the cases was 3 c.c. intramuscularly. Two of my patients presented such obvious arterial degeneration that some untoward result could have been expected if such were occasionally to follow the use of this blood coagulant. My brief experience with Koagamin indicated that it is a safe drug and that its use facilitates greatly the operation of a transurethral prostatic resection." Koagamin is made by the John A. Millar Company at 11 Hill St., Newark, N. J. It does not deteriorate if kept in a dark place. It furnishes increased blood viscosity. It is nontoxic. The dosage is 2 c.c. intramuscularly, one-half preoperative, followed by 2 c.c. intramuscularly immediately after the operation to prevent bleeding; in profuse hemorrhage 3 c.c. to 5 c.c. intravenously, followed by 2 c.c. intramuscularly within thirty minutes. Additional administrations of 6 c.c. in three divided doses at hourly intervals can be given if the hemorrhage persists; in capillary bleeding 2 c.c. intravenously, followed by 2 c.c. intramuscularly within an hour if necessary; in chronic blood dyscrasia 4 c.c. intravenously followed by a maintenance dose of 2 c.c. intramuscularly three times a week until symptoms have improved. For infants dosage should not exceed 4 c.c., with 2 c.c. intravenously and 2 c.c. intramuscularly in doses of not more than 1 c.c. at a given time. The contraindications are essential hypertension, peripheral vascular diseases, and diseases where there is a predisposition to thrombus.

Snake Venom.—Moccasin venom has been used successfully for the control of bleeding in a number of pathologic states, especially where the hemorrhagic tendency is due to capillary fragility, such as hemorrhagic purpura, etc.

This test consists of injections of 0.1 to 0.2 c.c. (1 to 2 hemorrhagic units) of standardized moccasin venom intradermally and reading the reactions in thirty minutes to one hour. A positive test is in the form of an ecchymosis at the injection site in one hour.

Peck states: "In preparing patients for tooth extractions with moccasin venom the initial injection in those of over 10 years of age is 0.4 c.c. of the 1:3000 dilution. The amount is rapidly increased in subsequent injections to 1 or 2 c.c. as a maximum single dose. The injections are given subcutaneously in the arm or thigh. Daily administrations of venom can be given. The duration of the treatment depends on the type of case treated. If sufficient time can be spared before the extraction takes place, the therapy is continued until the venom test becomes negative. In children, especially those under 5 years of age, the minimum amount injected is 0.2 c.c. of the 1:3000 dilution, and 1 c.c. as a maximum amount.

"A certain number of patients may develop a reaction of hypersensitivity to the snake venom in ten to fourteen days after the beginning of the treatment. The reaction is always local and is characterized by a hot tender swelling at the injection site. Desensitization is easily carried out. The injection after the reaction of hypersensitivity is reduced to 0.05 c.c. of a 1:3000 dilution, and subsequent doses are increased very slowly until 1 c.c. can be given again."

Winter states: "The use of snake venom in our experience has not been of any practical value in the control of bleeding in hemophiliacs."

Congo Red.—This is being used rather extensively, 10 c.c. for intravenous injection in prostatic surgery. Five to 10 c.c. of 1 per cent sterile aqueous solution of Congo red administered intravenously in acute hemorrhage has been recommended. It is stated that the blood platelets and the fibrinogen content are increased and the coagulation time reduced; with also an increase in the number of monocytes, which indicated a rather hyperfunctioning of the reticulo-endothelial system.

Vitamin K.—Vitamin K is a newly discovered factor which is essential to blood clotting. It is produced normally by bacteria in the lower intestinal tract, from which it is absorbed. When absorption is faulty, Vitamin K deficiency occurs, as absorption of the fat-soluble substance depends upon the bile. Obstructive jaundice will therefore produce Vitamin K deficiency, and this condition may be relieved by feeding Vitamin K and bile salts.

It is believed that Vitamin K is necessary in the normal synthesis of prothrombin in the body.

Dam[7] in his biochemical studies on chicks found that hemorrhage resulted when they were fed a fat-free diet. Dam suggested the name Vitamin K for this unknown substance.

The value of Vitamin K as a prophylactic and curative factor in hemorrhagic disease of the newborn is well known.

Blood Transfusion.—A transfusion is the transfer of blood from one person (the donor) to another person (the recipient). There are various indications for blood transfusion, no doubt the most important indication being acute anemia following a severe hemorrhage. It is also used in hemophilia and other conditions with lessened coagulability of the blood, in septicemia, gas poisoning (CO), acid intoxication, in malnutrition, shock, and in leucemia. It gives temporary relief in pernicious anemia. Before and after operations when the patient is in a weakened condition, blood transfusions may be indicated.

Certain preliminary tests should be performed with samples of the blood of both donor and recipient, to exclude the possibility of transmitting disease and to determine whether the blood of donor and recipient are incompatible. The serum of the blood of one person may cause agglutination or hemolysis of the red blood cells of the other person. A Wassermann or Kahn Test should be made to exclude the possibility of conveying syphilis. The conveyance of other diseases must also be considered, and therefore the donor must be very carefully examined.

There are four sharply differentiated blood groups and the blood of each person will fall into one or another of these four distinct groups. The transfer of blood which is incompatible may be attended with serious symptoms and hence it becomes necessary to know into what group each person falls. The Moss classification of blood types is used very extensively. Group I in the Moss classification corresponds to Group IV in the Jansky classification. According to the Moss classification the greatest number of persons fall into Group IV, next in Group II then into Groups I and III respectively. The blood should be selected from a donor whose blood belongs to the same group as does the blood of the recipient. In case of emergency a donor of the Moss Group IV may give blood to a person who belongs to any group, provided that the transfusion is slowly performed. If symptoms of incompatibility occur during transfusion, it should be stopped.

The amount of the blood to be given depends on the condition of the patient, the reason for the transfusion, and upon the donor. The usual dosage for adults varies from 500 to 1000 c.c., although in some cases it is advantageous to give 250 c.c. and repeat when necessary. In some cases small amounts, given as the condition demands, give better results than when large amounts are used. Blood transfusion possesses certain advantages over saline infusion, since it supplies nutritive material and oxyhemoglobin and does not leave the vessels as quickly as does saline solution to be excreted.

Methods of transfusion may be subdivided into direct and indirect. Unaltered whole blood or citrated blood may be transfused into the indirect method. In direct transfusion the blood is transferred directly from the vein of the donor to the vein of the recipient. It takes considerable time and skill on the part of the operator, and it is difficult to estimate the amount of blood transferred. In indirect transfusion of unaltered whole blood, special syringes are used to transport the blood from the donor to the recipient. No anticoagulant is added. This method requires at least three operators. The sodium citrate method consists in withdrawing the blood from the donor into a flask containing sodium citrate which prevents coagulation of the blood. Some authorities claim the anticoagulant increases the fragility of the red blood corpuscles. The latter method is used most extensively.

Intravenous Infusion.—It consists in the injection of a solution into a vein. It is used in hemorrhage and other conditions to restore the volume of blood immediately to normal. The solutions used are normal saline solution, Ringer's solution, Locke's solution, and Dawson's solution. To understand the effects of injection of these various solutions requires a knowledge of their chemical composition. There is considerable danger involved in intravenous infusion due to

injury to the vein followed by phlebitis, injection of bacteria into the blood stream, and of injection of air and foreign matter. Only distilled water should be used in making up these solutions. The temperature of the injected solution should be from 110° to 118° F. The procedure must be conducted with aseptic precautions.

Hypodermoclysis.—It is the injection of physiologic saline solution into the subcutaneous tissues. The solution may be injected beneath the skin of the abdomen, thighs, buttocks, or below the breast. The uses and effects of hypodermoclysis are much the same as in intravenous infusion. Both treatments are contraindicated in edema.

Natural Arrest of Hemorrhage.—Nature itself reacts to injury and loss of blood. An extensive hemorrhage tends to increase the coagulability of the remaining blood. The force of the blood current is reduced, and the blood is able to clot. When a small artery is cut, its muscular walls contract and there is a retraction of the vessel wall. Thrombosis occurs outside the vessel which impedes the flow of blood. When large blood vessels are injured, clots cannot form quickly because of the force of current, and a large amount of blood may be lost. When a clot has been formed in a vessel, organization occurs. The clot is replaced by fibrous tissue which permanently occludes the vessel. The blood-forming organs manufacture an increased number of cells to compensate for the loss of cells during hemorrhage.

Results of Hemorrhage.—It is obvious that if hemorrhage is not checked, the death of the patient will result. The immediate cause of death is cerebral anemia resulting from a sudden fall of the blood pressure due to loss of blood. Dyspnea due to defective aeration precedes death. Syncope, a condition of more or less complete unconsciousness due to anemia of the brain, results. There are various methods of relieving this cerebral anemia. A change in the position of the body and the introduction of saline solution or blood into the body may remedy the condition. The diet is of special importance in general anemia. Iron and arsenic are the drugs used in anemia. Iron aids the formation of hemoglobin, arsenic stimulates the bone marrow in the formation of red blood cells. The anemic patient should be given complete rest and freedom from mental and physical conditions which increase the strain on the heart. Diseases existent previous to hemorrhage at times take on renewed activity after hemorrhage. At this time patients are also more susceptible to infection, because of a lowering of the resistance of the body.

CASE HISTORIES

Mr. R. D., aged 18 years, a student, presented for removal of four impacted third molars on Aug. 30, 1939. The mandibular third molars showed definite evidence of bone resorption and infection. He was given a general anesthetic and all four of these teeth were removed at one time. He was hospitalized and got along very nicely.

At midnight on Sept. 4, 1939, he had severe bleeding in the right mandibular third molar, and it was packed with gauze. The following day he had bleeding on the opposite side. Both of these areas were controlled immediately. The right mandibular third molar area began to suppurate along the buccal incision. This was evacuated through the mouth, and he progressed very rapidly.

On Sept. 7 he had a severe hemorrhage. There was continuous oozing of blood for the next few days. We were able to control this by changing the dressing and using a tannic acid solution upon the dressing. On the right side of the face the infection again stirred up and it was necessary to make an extra-oral incision for drainage of pus. This went along nicely for two days and apparently cleared up, progressing very satisfactorily. His temperature went down to 99° F.; then suddenly he had bleeding from both sides. There was a gangrenous condition of the tooth sockets and surrounding tissue with a formation of a bulging mass of soft tissue with a hematogenous appearance in the region of the mandibular third molars. Both submaxillary glands were somewhat swollen. The lips swelled with an appearance of angioneurotic edema and the right side of his face immediately took on the appearance of a beginning erysipelas, with the leucocyte count around 20,000. His blood was Type III, and, since his mother's blood was found to have the same picture he was given a transfusion of 250 c.c. of her blood. The oral tissue immediately took on a better appearance. He was given 80 gr. of sulfanilamide in twenty-four hours, 15 gr. every four hours, using the neosulfanilamide. The tablet was sulfanilamide, 5 gr., sodium bicarbonate, 5 gr. The following day the dose was cut to 40 gr. and the next two days cut to 40 gr. He did not experience nausea, and there was no cyanosis. The gauze dressings were left out of the mouth; his mouth was irrigated frequently, alternating chlorazene solution and detoxol. He was given forced liquids and a liquid diet, and he gradually improved. The evening of the transfusion his temperature went up to 104° F. with a pulse of 138, at which time the sulfanilamide was started. The temperature went down the next morning to 99 and remained there.

He remained one week without further disturbance and was dismissed.

The significant thing about this case is that the patient developed bleeding as a result of lowered resistance. Swelling, edema, infection, gangrenous tissue, erysipelas, and bleeding were controlled entirely by blood transfusion and sulfanilamide. All dressings were removed; wounds were left alone and allowed to bleed, which continued about twenty-four hours after transfusion; and sloughing tissue was not disturbed.

Mr. P. N. G., aged 29 years, was first seen on Feb. 20, 1940, with a chief complaint of discomfort around the mandibular right third molar. X-ray examination showed this to be impacted and both maxillary third molars to be impacted. We discussed the advisability of removing all of these at one time.

This boy gave a history of having had his tonsils out six years previously at which time he had had rather profuse bleeding for a period of twelve hours. This was finally brought under control, however, without any unusual difficulty. He was given two blood transfusions to compensate for the blood he lost at that time. He had no history of bleeding before that time or since. Inasmuch as this was not an uncommon complication following tonsillectomy and in view of the negative history otherwise, we felt that it was probably not of much significance.

A bleeding and clotting time was taken, which was normal, but as an extra precaution he was put on dicalcium phosphate, grains one, t.i.d. He was also given six A, B, D, and G capsules daily and advised to drink lots of milk and take large amounts of orange juice. He returned on March 5, and under gas anesthesia, both maxillary third molars and the mandibular right third molar were removed. These were not difficult extractions. He was returned to his room following the operation and left the hospital in a few hours.

At 8 P.M. that evening I was called, and he was bleeding profusely from all of the sockets and had been for the past three hours. The sockets were all packed with bleeding solution but this did not help. Finally, after packing the sockets very tightly with iodoform gauze and Monsel's solution the bleeding stopped. The patient was kept in the office hospital over night. He had pain in the lower jaw which was brought under control by empirin. There was slight oozing throughout the night. The following morning there was no bleeding, and he was discharged.

On March 7 he was seen and as there was no bleeding the dressings were not disturbed.

On March 8 he came in in the morning bleeding from area number sixteen. This was brought under control with Monsel's solution and iodoform gauze. That night at 11:30 I was called to the office to see him at which time there was bleeding from areas numbers one and seventeen. It was finally stopped with iodoform gauze and Monsel's solution.

The following day there was no bleeding. He was given a gingival treatment and there was some swelling and pain in the region of the lower right jaw.

On March 10 there was oozing throughout the day which could be controlled somewhat by biting down on cotton.

On March 11 there was moderate bleeding from all of the sockets throughout the day, and he was admitted to our hospital. At 5:30 he was given 3 c.c. of coagulin Ciba, intravenously and 17 c.c. intramuscularly in the right buttock. There was slightly increased swelling and pain, requiring codeine, in the lower right socket. Bleeding continued throughout the night from the lower right socket. Apparently the coagulin had no effect. At midnight it was necessary to return to the office to repack the area with gauze and Monsel's solution. The bleeding was brought under control with pressure.

The following day there was slight oozing and at 10:15 A.M. on March 12, he was given 4 c.c. of Koagamin (Millar) intravenously. Codeine was continued for pain. At 8:00 P.M. he was given 5 c.c. of Koagamin (Millar) intravenously. At 1:00 A.M. he was still seeping steadily. He continued to bleed throughout the night, and at 8:00 A.M. the following morning there was still moderately severe bleeding, and there was increased swelling on the right side of the face.

He was admitted to Doctors Hospital on March 13 and given 500 c.c. of citrated blood. The transfusion seemed to improve him somewhat, and he looked better that afternoon but still there was some seepage. X-rays of the jaws were negative for any evidence of bony involvement.

On March 14 there was no bleeding.

On March 15 the dressings came loose and were changed followed by a very slight amount of bleeding. He was given another 500 c.c. of citrated blood

on March 15 and that afternoon the bleeding ceased. He was given two injections of 5 c.c. each of Koagamin (Millar) on March 15.

He was discharged from the hospital on March 16 and remained in bed for four days after he returned home. The dressings were removed only as the upper portion became loose. This was cut off and the part of the dressing that was still tight in the socket was not removed. He had periods of slight oozing after he returned home but they were very minor and he was given two additional intravenous injections of 5 c.c. of Koagamin after he returned home.

All of the tests made at the time he first entered our hospital were normal. They included the Wassermann, urinalysis, and complete blood count. The hemoglobin before the transfusion was 78 per cent and the red cell count was 4,000,000. The platelets count was 250,000.

The dressings were removed on March 23, 1940.

In summarizing this case it is difficult to determine the cause of the bleeding. His family history is negative and aside from the one previous attack of bleeding, when his tonsils were removed, this boy had had nothing similar. The platelets were 250,000, but this is not unusually low as they are normally between 300,000 and 450,000. There is the possibility of a vitamin deficiency in this case as this boy gave a very irregular dietary history with not much in the way of citrous fruits and fresh vegetables in his diet.

Apparently the most effective measure used in the control of the hemorrhage was the two transfusions of 500 c.c. of citrated blood. The effects of the Koagamin were problematic and the effects of the coagulin Ciba were nil as far as control of bleeding was concerned. The Monsel's solution apparently had a better local coagulation effect than the bleeding solution but it is less desirable from the standpoint of the disagreeable deposit it leaves in the mouth when it is used in excess.

Mr. H. A. S., aged 66 years, well developed, well built, florid appearance, presented to the Washington Sanitarium June 20, 1924, complaining of headaches, inability to sleep well, constipation, and nervousness. His physical examination was negative, but the blood test proved striking, showing a red cell count of 7,000,000 with the white cell count 20,000, with a color index of .97. Roentgenograms of mouth showed four mandibular teeth bad and extraction was advised. The mucous membranes were badly infected and there was gingival hemorrhage around the necks of the teeth. The condition was diagnosed as polycythemia, with an increase in white cell count as well as hemoglobin. At the present time he has had treatment for more than a year, which is constituted largely of phenylhydrazine, 1½ grains three times a day after meals, watching carefully for accumulative action after about a two-week period. His hemoglobin and red cells are about normal with white cells about 21,000. He seems to me to be making definite improvement.

Mrs. H. F., aged 48 years, was a housewife. She presented with symptoms of toxic absorption. The gums were inflamed and there was bleeding from most of the gingival spaces. X-rays showed twelve infected teeth. There was an eight million red blood cell count. The case was diagnosed as polycythemia and the teeth were removed very cautiously, one or two at a time over a long period of time. Hemorrhage was controlled with some difficulty. The only effective

means was to cauterize with diathermy. Other methods proved of little avail. This patient was observed over a period of four years and finally died, although the hemorrhage from tooth extraction was always controlled.

SUMMARY

1. A very careful clinical examination should be made, supplemented with general physical examination and laboratory tests, where necessary, in order to make a correct differential diagnosis.

2. Wherever there is a systemic disorder, the aid of the attending physician should be sought in order to correct any abnormality before surgery.

3. If bleeding or clotting time is at all abnormally high, an attempt should be made to correct this before surgery.

4. Where the patient gives a history of previous bleeding, careful attention to diagnosis and prevention should be carried out. Very little dependence should be placed upon the usual types of remedies, such as calcium lactate, calcium gluconate, gelatin, and many of the proprietary preparations.

5. Compound tannic acid solution is the best agent for local use in the mouth.

6. There seems to be much merit in the use of citrus fruit and juice to provide vitamin P with which to correct capillary fragility.

7. Vitamin K is beneficial in cases of biliary deficiencies and in hemorrhagic diseases of the newborn.

8. Koagamin has proved very beneficial in many cases.

9. The one great method upon which all surgeons depend, in the final analysis, is transfusion, which is prescribed 250 c.c. repeated where necessary and in some cases 500 c.c. This is usually a postoperative measure, but in extreme cases may be used as a preoperative measure.

REFERENCES

1. Agranat, A.: Congo Red as a Hemostatic, South African M. J. **10**: 332, 1936.
2. Almquist, H. J., Mecchi, E., and Klose, A. A.: Estimation of the Antihemorrhagic Vitamin, Biochem. J. **32**: 1897, 1938.
3. Birch, C. L.: Hemophilia and the Female Sex Hormone, J. A. M. A. **97**: 244, 1931.
4. Bruckner, V., and Szent-Gyorgyi, A.: Chemical Nature of Citrin, Nature, London **138**: 1057, 1936.
5. Chew, W. B., Stetson, R. P., et al.: Estrogenic, Luteal, and Gonadotropic Hormones in Hemophilia, Arch. Int. Med. **55**: 431, 1935.
6. Cook, Thomas J.: Blood Dyscrasias From a Dental Point of View, AM. J. ORTHODONTICS **24**: 467 and 580, 1938.
7. Dam, H.: Vitamin K, Ztschr. f. Vitaminforsch. **7**: 248, 1938-39.
8. Editorial: Hormones in Treatment of Hemophilia, J. A. M. A. **104**: 1422, 1935.
9. Epstine, Irwin A.: A Quantitative and Qualitative Considerative of the Blood Cells in Normal Blood, J. A. D. A. **16**: 1064, 1929.
10. Fantus, Bernard, and Dyniewicz, H. A.: Compound Solution of Tannic Acid, J. A. M. A. **109**: 200, 1937.
11. Goadby, K.: J. A. D. A. **9**: 373, 504, 1922.
12. Graves, K. C., and Kickham, C. J. E.: Congo Red for Control of Bleeding, New Eng. J. M. **214**: 782, 1936.
13. Lajos, S.: Clinical Experiences With Citrin (Vitamin P), Klin. Wchnschr. **16**: 1615, 1937.
14. Lotze, H.: Researches on Vitamin P, Ztschr. f. d. ges. exper. Med. **102**: 697, 1938.
15. Lotze, H.: A Critical Note on "Vitamin P," Deutsche med. Wchnschr. **64**: 477, 1938.
16. Mead, Sterling V.: Diseases of the Mouth, St. Louis, 1940, The C. V. Mosby Company.
17. Milliken, L. F.: The Use of a New Blood Coagulant in Transurethral Prostatic Resection, J. Urology **42**: 75, 1939.
18. Moll, T.: Vitamin P, Klin. Wchnschr. **16**: 1653, 1927.

19. Moral, Hans: Einfuhrung in die Klinik der Zahn und Mund Krankheiten, Leipsic, F. C. W. Vogel, 1922.
20. Peck, Saml: The Use of Snake Venom in Dental Practice, Arch. Chem. and Oral Path. **1**: 5, 1937.
21. Scarborough, Harold: Vitamin P, Clinical Laboratories, Royal Infirmary, Edinburgh, 1400-1407, July, 1939.
Scarborough, H., and Stewart, C. P.: Effect of Hesperidin (Vitamin P) on Capillary Fragility, Lancet **2**: 610, 1938.
22. Schonheyder, H. D. F., and Tage-Hansen, E.: Studies on the Mode of Action of Vitamin K, Biochem. J. **30**: 1075, 1936.
23. Schumann, E. A.: The Treatment of Postpartum Hemorrhage, Am. J. Obst. and Gynec. December, 1939.
Idem: The Treatment of Uterine Bleeding, Am. J. Surgery, at press.
Idem: Newer Concepts of Blood Coagulation and the Control of Hemorrhage, Am. J. Obst. and Gynec., December, 1939.
24. Steinberg, A., and Brown, W. R.: A New Concept Regarding the Mechanism of Clotting and the Control of Hemorrhage, Am. J. Physiology **126**: 638, 1939.
25. Szent-Gyorgyi, A.: On Vitamin P, Bull. Acad. de med. de Roumanie **2**: 17, 1937.
Idem: Methods for Preparation of Citrin, Ztschr. f. physiol. Chem. **255**: 126, 1938.
26. Tainter, M. L., and Throndson, A. H.: Hemostatic Effects of Administration of Calcium Viosterol, Styptysate, and Ceanothyn by Mouth, J. A. D. A. **25**: 638, 1938.
27. Throndson, A. H., and Richardson, A. P.: Alleged Hemostatic Action of Gelatin, Coagulin, Fibrogen and Histidine Administered by Mouth, J. A. D. A. **26**: 420, 1939.
28. Zacho, C. E.: Influence of Ascorbic Acid and of Citrin on the Capillary Resistance of Guinea Pigs, Acta path. et microbiol. scand. **16**: 144, 1939.

Case Reports

Cases of fibro-osteomas, which often involve enlargement and deformity of the jaw, are fairly common tumors. They are benign and should be carefully differentiated from similar tumors of the jaws which are malignant.

Case reports for this section of the JOURNAL should be sent to Dr. Kurt H. Thoma, 53 Bay State Road, Boston, Mass.

CASE REPORT NO. 43

FIBRO-OSTEOMA (OSTEITIS FIBROSA LOCALIZATA) CLINICALLY AND RADIOGRAPHICALLY RESEMBLING EWING'S TUMOR

MAX H. JACOBS, M.D., D.M.D., BOSTON, MASS.

OSTEITIS fibrosa may clinically and radiographically simulate one of a number of bone growths of the jaws. The histopathology of osteitis fibrosa varies quantitatively in its distribution of converted bone. Bone marrow may be replaced by a greater or lesser amount of fibrous tissue. For this reason a varied terminology has arisen.

Fruedi[1] has termed these bony enlargements, osteofibromas. Phemister and Grimson[2] use the term, fibrous osteoma. Thoma[3] uses a terminology based upon the proportion of soft and hard tissue present, recognizing different stages in the tumor formation. His classification is, "(1) the fibromatous stage (central fibroma), (2) the ossifying stage (ossifying fibroma), and (3) fibro-osteoma: (a) fibro-osteoid osteoma and (b) sclerosing fibro-osteoma."

Because of these varying stages in which one of these tumors may occur, preoperative diagnosis may at times be difficult, and actual diagnosis can be made only following biopsy or operation.

CASE HISTORY

R. F., a female, aged 12 years, presented herself with an enlargement of the left side of the mandible. Three years previously she had fallen on the left side of her face. About three months previously, her mother noticed that the left side of the mandible was much larger than the right. The mother felt that at times the swelling would decrease in size. On occasions the patient was unable to open her mouth widely. There had been no pain associated with the swelling until about a month before the patient came for consultation, when she developed German measles. At this time the swelling reached its largest size and had not receded.

Examination disclosed a distinct enlargement of the mandible extending from the corner of the left lip to the angle of the jaw. On palpation, the mandible itself was found to be markedly thickened buccolingually. A rounded mass which seemed to be continuous with the lower border of the jaw projected into the neck. This mass was not movable, but the skin was movable over the mass.

It had the consistency of bone and not that of enlarged glands. There were no palpable submandibular glands. Buccally, the greatest bulge was opposite the second molar area.

Electro vitality tests disclosed all of the mandibular left teeth were vital.

X-ray Examination.—X-ray examination revealed a marked radiopaque density of the mandible extending from the first premolar to the third molar area. The roots of the second molar were not fully formed, and there was an irregular radiolucent area below and around these roots. Below the mandible, and apparently attached to it, was a somewhat radiolucent circumscribed mass (Fig. 1).

X-rays of the long bones were essentially negative, but seemed to border on the osteoporotic. The vertebrae were negative.

The blood calcium was 11.3 mg. per 100 c.c.., and the blood phosphorus, 3.5 mg. per 100 c.c. The blood counts and differential were within normal limits. The urine was negative and contained no Bence-Jones bodies. A Wassermann test was negative.

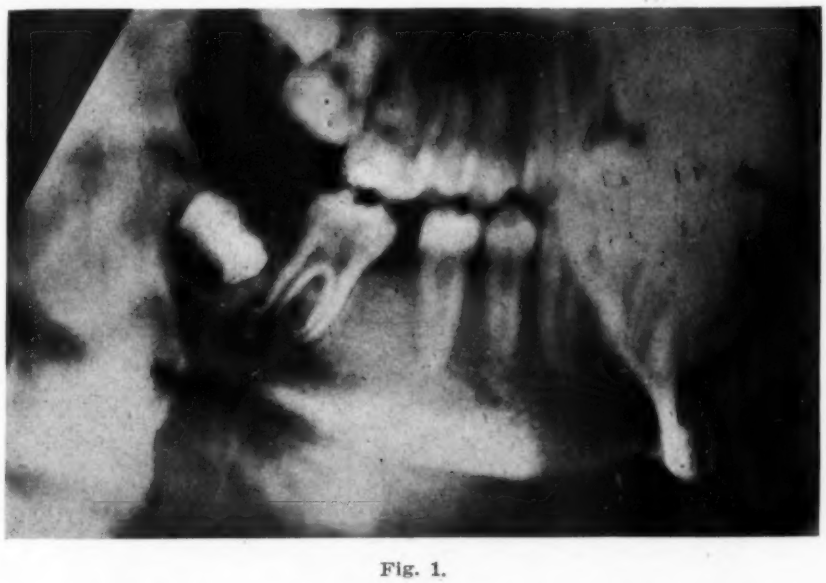

Fig. 1.

In evaluating the evidence present necessary to make a preoperative diagnosis, cyst, osteogenic sarcoma, and osteomyelitis could be ruled out. Ewing's tumor (endothelial myeloma) and osteitis fibrosa remained to be considered.

Both conditions occur in young life. Both are said to be caused by trauma, and in the jaws occur as solitary lesions. Pain is present more often in Ewing's tumor than in osteitis fibrosa.

X-ray evidence in favor of Ewing's tumor was the radiolucency in the central mass of the mandible denoting bone destruction. This was characteristic of Ewing's tumor. The large circumscribed mass attached to and under the jaw could well have been part of the picture, because many times Ewing's tumor has its start subperiosteally, entering the bone centrally and proliferating through the periosteum externally.

On the other hand, the fine trabeculations or stippling seen in the x-ray as a radiopaque area were consistent with that seen in the fibro-osteoid osteoma.

Biopsy.—A biopsy was performed. An incision was made extending from the anterior border of the vertical ramus along the ridge to the distal surface of the second molar. The incision was continued along the buccal surfaces of the gingival tissues of the teeth through the interdental papillae as far as the canine. The mucoperiosteum was reflected and retracted. It was interesting to note that the periosteum was intact. The buccal surface of the mandible from the distal of the second molar to the first premolar was composed of a reddish honeycomb type of bone which cut very easily and was exceptionally spongy. There appeared to be no cortical bone, and the medullary portion was entered easily.

Retracting the tissues below the border of the mandible, the spongy bone was seen to continue in the form of a mass below the jaw into the neck. This bone was softer in consistency than the bone overlying the mandible, but both had the same characteristics.

There was no excessive bleeding during the removal of sufficient bone for examination.

Microscopic Examination.—Microscopic examination of five slides showed the bone to consist of a fibrous tissue stroma, somewhat edematous and surrounded in general with osteoblasts. Portions of fibrous tissue, dense in nature, were associated with a scattering of round cells, and a few polymorphonuclears were seen.

Fig. 2.

In places there seemed to be a definite transition from bone to osteoid tissue. The fibrous tissue entirely replacing the marrow was composed of a very loosely arranged tissue with a fairly rich blood supply consisting of small capillaries. In the loose net of collagenous fibers were scattered fusiform well-developed fibroblasts. There was no evidence of malignancy anywhere, and there were no excessively cellular areas (Fig. 2).

Diagnosis.—Osteitis Fibrosa.

Comment.—Apparently this is one of those rare cases of osteitis fibrosa which penetrated through the cortex to continue its growth by expansion into the soft tissues.

REFERENCES

1. Fruedi, Anna: A Study of So-Called Osteofibromas of the Maxilla, Dental Cosmos **77**: 999, 1935.
2. Phemister, D. B., and Grimson, K. S.: Fibrous Osteoma of the Jaws, Ann. Surg. **105**: 564, 1937.
3. Thoma, Kurt H.: Central Osteomas and Cementomas: Diagnosis and Treatment, J. A. D. A. and Dental Cosmos **25**: 750, 1938.

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Anatomic Principles as Applied to Dentistry: By T. Seward, D.D.S., Melbourne, Australia, *J. A. D. A.* **27**: 1021, July, 1940.

Two characteristics of the muscles moving the jaws are (1) the comparative weakness of those that open the jaws and (2) the extraordinary power of those that close the jaws.

Most textbooks state that the human jaws are opened by the external pterygoids and the hyoid muscles. It seems likely that the jaws are opened by the external pterygoids, but for two reasons it is most unlikely that the hyoids play any part in the action: Man can open his mouth against a comparatively great pressure and, at the same time, swallow. It is impossible for the hyoid muscles to take part in two unrelated simultaneous movements, i.e., elevating the hyoid bone and depressing the mandible.

An examination of the muscles of mastication in man reveals a large external pterygoid. By pulling the condyle forward on the eminentia articularis, it is possible for the external pterygoid to open the jaws, but, in the carnivora, the condyle cannot move out of the glenoid fossa. Therefore, either the function of the external pterygoid differs with the species or our interpretation of the muscular action is wrong.

The power available for closing the jaws is enormous. Circus girls have supported the weight of more than six men with their teeth. All the muscles of mastication take part in the closing of the jaws, and the individual muscles of the group are developed according to the character and the requirements of the animal.

The *masseter* is a very powerful muscle attached to the mandible. Its origin is given as the zygomatic arch. Attached to the upper border of the zygomatic arch and continued around the skull is the galea aponeurotica, a tough, inelastic, tight-fitting skullcap. When the masseters contract, the pull passes through the zygomatic arches, to be transmitted by the galea aponeurotica evenly over the entire vault of the skull.

The *action of the temporal muscle* is not as described in the textbooks; it is most easily understood by referring to the carnivora. When the temporal muscle contracts and pulls the high coronoid process backward, the anterior part of the mandible is raised, and the posterior part, including the condyle, is lowered. If the posterior part of the mandible were not supported, the tem-

poral muscle would put a severe strain on the ligaments binding the condyle in the glenoid fossa. It is here that the action of the masseter is intimately associated with the temporal, for, having as its base the whole vault of the skull, it can raise the posterior part of the mandible, harmoniously supporting the joint against any contraction of the temporal. Strong temporal muscles are always associated with strong masseter muscles.

The teeth of man are intermediate between those of the carnivora and herbivora. The cheek teeth meet almost end on, and, as in the carnivora, there are incisors and canines. The zygomatic arches are close to, and nearly parallel with, the sides of the skull. The temporal, masseter, and internal pterygoid muscles are more evenly balanced, while the condyle and the glenoid fossa resemble those of both the carnivora and the herbivora.

The condyle and the glenoid fossa have something in common with the muscles of mastication; whenever power and precise movement are necessary in the anterior part of the mouth, we find a powerful temporal muscle and a highly specialized condyle.

If the jaws were slung by muscles and ligaments alone (both condyles are sometimes surgically removed in man), power and rapidity of movement would not be hindered; immediately, however, when the condyle is added to the attachment, the most precise action is possible. This, then, is the explanation of the closed, accurately fitting joint of the carnivora and the open one observed in the omnivora, where precise movement is not necessary.

The characteristics common to all skulls are:

1. There are always two hard parts, vault and mandible, that are separated by a weak part, face bones.

2. The mandible is a completely separate bone.

3. The upper jaw is immovable and is attached to the skull by columns of bone that pass through the weak face bones. By attaching the masseter to the zygomatic arch, Nature is able to change the direction of the line of action of this muscle to serve best her varied tooth patterns. With further binding and fixing of the skull to the mandible, the action of all the muscles of mastication is made more exact and more certain; as the lower jaw presses against the upper, the skull is caught firmly in this galea-masseter press, and, by this process, whatever upward force is used is equalized and dispersed through the vault of the skull.

The roots of the upper molars and premolars are disposed in two rows, buccal and lingual; the roots of the lower molars and premolars are always in a single row, anteroposteriorly.

From infancy, the skull is growing in all directions and continues growth until the last molar tooth erupts and takes its position, at which time the spheno-occipital suture, the last in the skull to do so, unites.

Although the sutures are observed to close at certain times, such as the second, twelfth, or between the eighteenth and the twenty-fifth years of life, it is much more important that those times coincide with the eruption of particular teeth. So, instead of teaching that the spheno-occipital suture, for in-

stance, closes between the eighteenth and twenty-fifth years, it would be more exact and more informative to describe the time as synchronous with the eruption of the last molar tooth.

When the first molar takes its place in the dental arch, the second permanent molar is forming, and, in developing, it pushes that part of the mandible anterior to itself forward, just as a growing plant will push rocks out of its way. If the roots of the first permanent molar have moved forward as Nature intended, the second and third molars will develop normally, but, if the weight of mastication is on the anterior half of the first molar, it is almost impossible for the posterior molars to develop and erupt normally. Man is the only living primate that has its teeth in a continuous series, and it is one of his distinctions that there are no gaps between them (Wood Jones).

An examination of the human skull will reveal that the long axes of the strong parts, vault and mandible, are not parallel, but approach each other posteriorly and diverge anteriorly. When the skull is squeezed in the galea-masseter press by the muscles of mastication, the face bones will take the line of least resistance and drift forward.

In function, the stress of mastication passing through the posterior half of the lower molars tends to rotate their large roots forward in the body of the mandible and promote the forward growth of bone. The molars, in developing, will naturally require more room in the body of the bone, and, in erupting, will push forward the teeth anterior to themselves.

By designing the upper teeth to overhang the lower, Nature still further insures the growth of the face bones. The strong lower jaw aids in growth and development of the nose and accessory sinuses. In architecture, columns, beams, and flying buttresses are used to support weight, or stresses pass through them to be dispersed elsewhere. In the skull, Nature uses the same devices, allowing the forces excited by the lower jaw to be transmitted through the weak face bones and dispersed through the skull.

Connecting the upper denture with the skull are three main columns of bone: (1) the anterior orbital column, (2) the posterior orbital column, and (3) the pterygoid column.

Acting as crossbeams and buttresses are, among other structures, the hard palate, walls of the orbit, zygomatic arches, plate bones, lesser wings of the sphenoid.

The skull is squeezed down onto the mandible by muscles of mastication acting through the galea-masseter press. The whole vertical force is received by the upper teeth, whose roots, being widely spread in two rows, are best designed to diffuse force through the bony columns of the skull. The lower teeth, whose roots are in a single row, are best designed to deliver force, as is the hammer that strikes the anvil.

When an accident occurs, Nature still further modifies her architectural plan, as follows, to serve the individual best. Loss of space due to shortening of the line of occlusion may be caused by (1) proximal caries or (2) loss of teeth.

There may be loss of resistance to the muscles of mastication. If the large first molars are lost, the resistance to the muscles will be weakened. Consequently, when the skull is squeezed down onto the mandible, vertical height will be lost, and a closed bite will result.

When the jaws close normally, the stress of mastication will pass almost vertically upward through the face bones and downward through the mandible. Under these conditions, the whole anatomic arrangement will promote normal growth of the face. If, for any reason, on eruption the first molars receive the weight of mastication on the anterior half instead of on the posterior half, their large roots will be prevented from rotating forward in the body of the mandible. This will, first, impede growth between the molar and the ramus, and, second, change the direction of the line of stress from mastication. Normal growth and development of the nose and accessory sinuses will thus be prevented. The mandible will flow forward, and a large chin will result. (This is especially true when maxillary molars are lost.—J. A. S.)

Aspects of Prevention of Malocclusion: By G. M. Anderson, D.D.S., Univ. of Maryland, Baltimore, *New York J. Den.* **10**: 215, 1940.

Anderson discusses preventive measures conducive to normal occlusal development of the teeth and the dental arches. He stresses the moral, which is to say, the professional, obligation of the dentist who must exercise his good judgment in evaluating articles on dental subjects. Many categoric statements made by dentists fall of their own contradictory evidence when examined. For example, there are Kronfeld and Logan's statements that the maxillary lateral at birth lies lingual to the deciduous central and cuspid; that these teeth, through jaw growth, move apart to allow the deciduous lateral to come into normal alignment with its neighboring teeth before eruption. In his experience that is not a normal occurrence, but rather presages malocclusion because of malposition of the tooth buds.

The so-called submerged deciduous tooth, which on clinical examination shows the permanent tooth crowns to have passed by the retained deciduous tooth, we are reminded, is not a case of submergence at all, but is brought about by ankylosis of the spongiosa of the jaw with the honeycombed absorbing deciduous tooth root, when the processes of absorption slow down for whatever the reason, the osteoblasts becoming active as the osteoclasts cease their activity. The growth of the maxilla and the alveolar process and the continuous eruption of the permanent teeth allow the condition to become progressively worse until the deciduous tooth is gradually overtaken entirely, giving it the appearance of being impacted.

This reviewer has found such conditions in children who were otherwise normal in every respect. Treatment consists in exposing and extracting the offending deciduous tooth. If the adjacent teeth are inclined over the ankylosed deciduous tooth, a space-opening device should be employed to re-establish the mesiodistal space prior to the extraction. The opening of the space may of itself stimulate the eruption of the deciduous tooth. In any event, the tooth can be easily extracted.

Periodic roentgenographic examination is of value in determining the presence of caries, as well as in noting the absorption of the deciduous roots and the presence, rate of development, and location of their permanent successors. It seems strange to be told at this late date that we should not make the mistake of "taking it for granted, that if you cannot see a cavity, it does not exist." Nevertheless, Anderson shows good reason why his advice is still pertinent.

Consideration is given by the author to loss of first permanent molars in early life and the disturbances in occlusion and increase in caries seen in these mouths. "It is bad enough," says Anderson, "through uncontrollable circumstances, for the molar to be lost, but is infinitely worse to be a party to unnecessary disorganization of the whole denture" by allowing uncontrolled drifting of the adjacent teeth.

The canine area is a region peculiarly susceptible to malocclusion. Any sudden movement or inclination of the lateral incisor prior to eruption of the permanent canines, especially if unilateral, should lead to suspicion of ectopic eruption, cysts, or other interferences. Unusually large spaces between deciduous laterals and canines may be due to erupting permanent canines mesial to the deciduous ones, with failure of the roots of the deciduous canines to become absorbed. In these cases, if allowed to continue, a malocclusion involving protrusion of the anterior teeth results. If the lower lip is held under the maxillary teeth, a pernicious malocclusion may result.

Bony Growths on the Jaw: Reprinted from *Science*, Aug. 2, 1940.

Dr. Aleš Hrdlička, of the Smithsonian Institution, has been making a study of about 5,000 lower jawbones of the collection at the institution of ancient and modern skulls, and finds bony outgrowths from the inner jaw surfaces fairly frequent, and widespread in both space and time. Some of these growths are tuberculate ("lumpy"); others are ridges. They are almost like bony tumors, except that the bone appears to be strong and perfectly healthy.

The first suggestion, that these growths represent an evolutionary "throwback," is set aside because ancient human and prehuman skulls do not show them. They are not found on Neanderthal jaws or on the jaws of fossil or existing great apes. They appear only on jawbones of modern man, and are slightly more frequent in men than women.

The most plausible explanation is that the growths are responses to muscular pull on the bones, due to chewing hard on tough, resistant foods. Especially significant is their relatively high frequency among the Eskimos. Also, they were more common on the jaws of early Norse settlers in Iceland and Greenland than they were in the Scandinavian homeland.

Asked about possibilities of results from the modern habit of gum-chewing, Dr. Hrdlička stated, "That question may not be as frivolous as it sounds. True, gum isn't tough, and it requires very little exertion to chew it. But the advanced gum addict chews and chews and chews, hours on end sometimes, so that the sum total of exertion on the part of the jaw muscles may easily amount to as much as a smaller number of harder bites on a tougher substance.

Interrelation Between the Vitamin B Complex and the Anterior Lobe of the Pituitary Gland: By D. C. Sutton and J. Ashworth, *J. Lab. & Clin. Med.* **25**: 1188-1192, 1940.

Despite the demonstrated specificity of nicotinic acid in the treatment of pellagra, some patients fail to respond to large doses of this drug, even when it is combined with liver extract and an adequate diet. The authors previously reported (*J. Lab. & Clin. Med.* **25**: 848, 1940) two patients with cachexia due to deficiency of the anterior lobe of the pituitary gland in whom pellagrous lesions improved upon the administration of polyansyn. Because of this experience, the authors decided to employ polyansyn (extract of anterior pituitary) in several patients presenting glossitis, cheilosis, as well as other clinical findings suggestive of pellagra. All of the patients had failed to respond to the usual treatment of pellagra, i.e., adequate diet, nicotinic acid, riboflavin, and parenteral liver. However, upon the daily administration of from 1 to 2 c.c. of polyansyn intramuscularly, the oral lesions cleared up promptly, and the patients were restored to health in a short time. From the experimental and clinical observations of Brennemann, Sommer, Hundhauser, and others, it appears that vitamin deficiency or partial starvation may cause a decrease of the hormones of the anterior lobe of the pituitary. This may serve to explain the good results obtained by the use of anterior pituitary extract in patients who had failed previously to respond to the accepted antipellagra treatment.

Current Treatment of Cancer of the Lip: By V. P. Blair and L. T. Byars, *Surgery* **8**: 340-352, 1940.

The authors take issue with the recent tendency to focus attention upon the simplified treatment of the primary lesion in cancer of the lip, rather than upon the cure of the patient. Although there probably is a cancer-free interval of varying length between maturity of a primary squamous epithelioma and the occurrence of regional gland contamination, we have no clinical means of recognizing the termination of this period. In cases under the observation of the authors, this period has varied from several months to thirteen years, glandular metastases having made their appearance as long as thirteen years after an apparently complete removal of the primary lesion. Blair and Byars favor prophylactic removal of the regional lymph nodes because of the difficulty and undependability of a satisfactory follow-up observation. However, since prophylactic removal of the regional lymph nodes is not always feasible or desirable, the authors feel that adequate irradiation should be administered in order to eradicate the primary lesion. This treatment should be instituted as early as possible.

Harry A. Salzmann, M.D.

Editorial

Future Research

War times are bringing many things forward into sharper focus. Research is one of these. It has long been realized by the medical profession that research is the fountainhead from which springs much of medicine's great advance, and that research has made modern scientific medicine possible cannot be disputed. Research started the chain of events that have brought about the relief of human ills impossible of accomplishment during the last World War.

In fact, it was in June, 1916, that the Research Institute of the National Dental Association was incorporated. At that time it occupied two large residences on Euclid Avenue in Cleveland. The first report issued in that year is interesting now, because it included the problems already begun, or contemplated as being important and worthy of original research. Among those listed were systemic diseases resulting from oral infections, joint diseases, iritis, nephritis, gastric and duodenal ulcer, glandular infections, pneumonia, pyorrhea and dental caries, and brown stain of teeth. The report further listed a substitute for platinum (dentists at that time were using about one-third of the world's production of platinum, valued at about $2,500,000 annually), root canal filling material, oral bacteriology, salivary disorders and facial deformities and their relation to the ductless glands. Had the dental profession been endowed with the grants and leadership provided the medical profession, it is reasonable to suppose that the above problems, as listed in 1916, would have been advanced much farther in their solution than most of them are today.

Notwithstanding the handicap of the lack of grants, the dental profession has made a highly creditable showing in the solution of some of its problems by the initiative of private practitioners, and the universities, usually working on their own resources.

In the orthodontic field alone, to mention only one instance, is McKay's fine original work on brown stain and mottled enamel in children. The research on this problem has made it possible for children to grow to manhood and womanhood with white, shapely teeth, which otherwise would have been unsightly brown, irregular, and mottled, as was formerly the case in certain geographic districts.

The significance and importance of that piece of research alone, are little understood by the layman. If, for instance, you should be able to take a Gallup poll of the public reaction to the question, "What are the cause and cure of brown stain and mottled enamel on children's teeth?" you no doubt would find that on account of the rugged individualism of sales-conscious radio announcers, the predominating reply would be "lack of iridium," or lack of some other miracle-worker incorporated in one dentrifice or another.

Former President Hoover now points out that the most important link in the solution of present-day social problems, and about the only hope, in fact, for a satisfactory breakdown of the problem, is research.

Research in dentistry, in like manner, is destined for far greater things in the future. Also important to the progress of research in dentistry is honest publicity directed for public information, sans the dramatics and fanfare that, unfortunately, have been responsible for much of the breakdown of the dental profession's confidence in the word research. The word has been exploited, dramatized, used in all kinds of sales and promotion campaigns; notwithstanding, it is still the key word to progress in all modern activities and its prestige should be more carefully preserved. The wisdom of the ant in keeping himself busy, coupled with research to find out new things, will be responsible for much of the progress of dentistry in the future, as it will in all branches and departments of human activity.

H. C. P.

Feature

Orthodontic Office

For the reason that offices are occasionally designed, constructed, and used exclusively for some particular department of practice, usually oral surgery or orthodontics, it is thought that those engaged in these specialties are interested in the design of offices that reflect new ideas adapted to such practice.

Such an office is the recently completed one of Dr. W. A. Giblin of Montclair, N. J. It is located in a remodeled house in a suburban community and is designed for the purpose of the practice of orthodontics.

The two illustrations herewith are of the waiting room and one of the operating rooms. They show some of the features adapted to this office, and a description of some of them, as worked out for orthodontic practice, follows.

EDITOR.

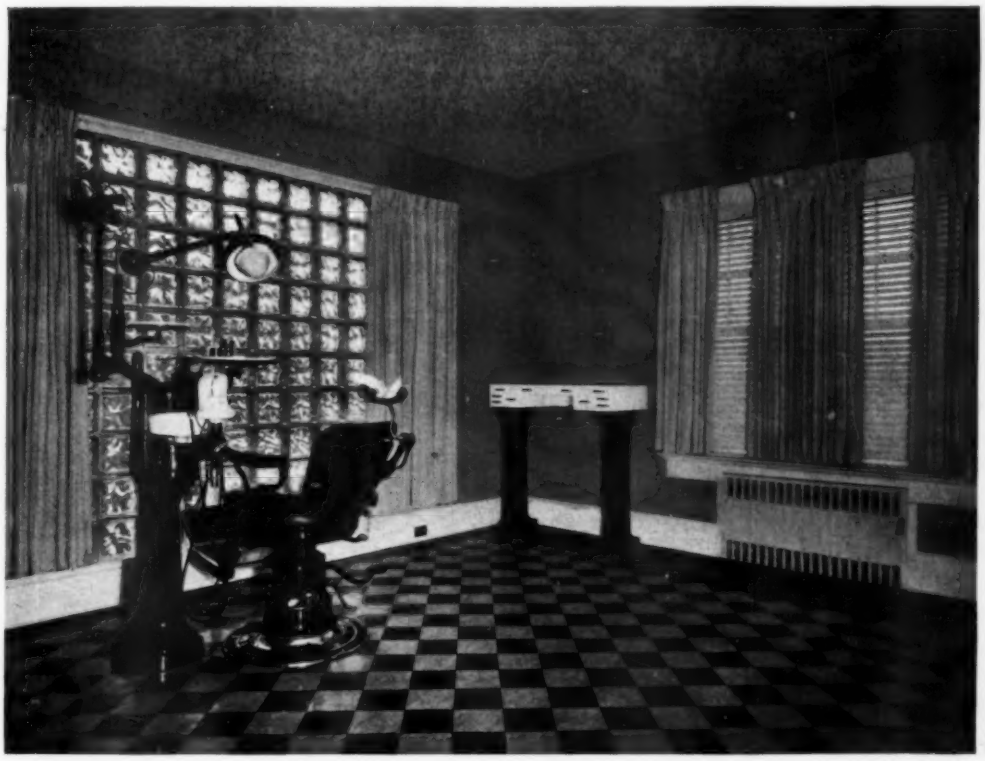
The reception room was planned to obtain maximum seating space without overcrowding, also to make it comfortable and pleasant. The wallpaper was selected to carry out the color scheme. Two specially built wall sofas covered in brown, with table and lamp between, take up the length of one side wall. One armchair in soft green stands by itself, also one straight-back chair with a seat cover of the same color. Particular attention was shown

View of the reception room.

for the smaller children in the selection of the low seat and chair covered in leather, with table and junior floor lamp, arranged in one corner of the room. At the opposite end are comfortable sectional chairs of striped fabric, with a corner table and lamp. All tables and woodwork, including cornices, are mahogany. A cabinet of the same kind of wood stands between two windows with a frameless mirror above. Floor length draperies are of green mohair, and the entire floor is carpeted in tan broadloom.

Blue was chosen for the walls of operating room No. 1 which faces south and east. The ceiling and woodwork are off-white. A marbleized linoleum with a black border blends with the wall coloring.

Operating room No. 2, facing north and east, is painted a rose tone with off-white woodwork and ceiling. The north wall of this room is almost entirely of glass block, giving an abundance of light at all times. Marbleized block linoleum covers the floor. Cream-colored casement curtains, sill length, are used in both operating rooms, while a draw curtain of the same material is used with the glass block to give a more softening appearance. There is cross ventilation in every room, and light is obtained from center ceiling fixtures and a unit light. This, with sufficient windows, has been found most satisfactory.



View of operating room No. 2.

The office is paneled in pine with executive and secretary's desk and chairs finished in maple. One armchair and one club chair are covered in dark green leather. Draperies are natural colored linen with embroidered diagonal stripe in shades of rust, green, and amber. Cornices are stained in mellowed tone to blend with the paneling. A filing cabinet is also painted to match. The floor is covered with jaspé linoleum in brown, with a plain border.

News and Notes

A. D. A. Convention Ends Sessions in Cleveland

The American Dental Association adjourned its eighty-second annual session in Cleveland, September 13, after extensive discussion of plans for national defense and after approving an annual increase of two dollars in dues.

Dr. Oren A. Oliver, an orthodontist, of Nashville, Tenn., was named president-elect of the Association for the coming year. Dr. Wilfred Robinson assumed the office of president at the close of the meeting to succeed Dr. Arthur H. Merritt, of New York.

The other officers elected were: James V. Gentilly of Cleveland, first vice-president; S. Blair Luckie of Chester, Pa., second vice-president; and Frank C. Cady of Washington, D. C., senior dental surgeon in the United States Public Health Service, third vice-president. R. H. Volland of Iowa City, Iowa, was re-elected treasurer, and Harry B. Pinney was re-elected secretary. Philip E. Adams of Boston was re-elected trustee. Other Trustees chosen are: Howard E. Summers, Huntington, W. Va.; Carlos H. Schott, Cincinnati, and H. B. Washburn, St. Paul, Minn.

Houston, Texas, was selected for next year's convention city.

The centennial convention brought together in Cleveland almost 9,000 members of the profession, in a meeting successful from every point of view. The convention's dominant theme, recurrent in the addresses of the Association's officers and distinguished guest speakers, was national defense and the dental profession's special role in it.

This theme found its first major expression, perhaps, in the report of the Committee on Economics to the House of Delegates. The report presented preliminary figures from a nation-wide dental health census, conducted by the Committee on Economics, showing that, in the words of C. Willard Camalier of Washington, D. C., chairman of the national defense committee, the nation, from a dental standpoint, "is woefully unprepared to meet the exacting requirements in industry and the military services in a time of possible national emergency."

Raymond M. Walls, of Bethlehem, Pa., concurred in this interpretation of the figures. Dr. Walls, as chairman of the Committee on Economics, supervised the census and presented the report to the House of Delegates.

The census, supervised by Dr. Raymond M. Walls of Bethlehem, Pa., the first survey of its kind ever attempted, was designed to secure an adequate cross section of the population's dental needs. Thirteen thousand dentists throughout the country received 65,000 questionnaires.

Dr. Walls and Dr. Camalier made it clear that their conclusions did not apply solely to the needs of those subject to military service, but also to the needs of the civilian population. Explaining that final computation of the census figures would not be completed for several weeks, Dr. Walls released the following partial results:

Average man in the United States between 20 and 44, inclusive, needs: 10.4 hours of dental service for 5.1 fillings, 2.1 extractions, 0.38 fixed bridges, 0.12 removable bridges, 0.18 partial dentures, 0.08 upper dentures, and 0.05 lower dentures.

In the part of this group aged 20 to 24, needs are: 6.5 fillings, 1.8 extractions, 0.34 fixed bridges, 0.11 removable bridges, 0.12 partial dentures, 0.03 upper dentures, and 0.01 lower dentures.

Aged 30 to 34, needs are: 5.5 fillings, 1.8 extractions, 0.33 fixed bridges, 0.16 removable bridges, 0.21 partial dentures, 0.05 upper dentures, and 0.01 lower dentures.

Aged 40 to 44, needs are: 3.6 fillings, 3.1 extractions, 0.34 fixed bridges, 0.15 removable bridges, 0.29 partial dentures, 0.18 upper dentures, and 0.14 lower dentures.

Commenting on the importance of the census, Dr. Walls pointed out that dental defects ranked second among the causes of military rejections in the United States in the First World War.

Dental Research Bill Passed by Senate

On Sept. 12, Senate bill 3607, which authorizes the Public Health Service to conduct researches relating to the cause, diagnosis, and treatment of dental diseases and appropriates $75,000 for this purpose, was passed by the Senate without amendment. The bill was introduced in the Senate on March 18 by Senator James Murray of Montana, and was reported favorably by the Committee on Education and Labor on April 26. The bill is now before the House of Representatives for its approval and has been referred to the committee on Interstate and Foreign Commerce.

Notes of Interest

Dr. Helen A. Gough announces the removal of her office to the Medical Arts Building, 142 Joralemon Street, Brooklyn, N. Y. Practice limited to orthodontics.

Dr. John M. Marré announces the removal of his offices for the practice of exodontics and oral surgery, to Suite 601-2-3 Missouri Theatre Building, St. Louis, Mo.

Dr. Harold E. Leslie announces the opening of his office for the exclusive practice of orthodontics, 345 Bloor Street West, Toronto, Ontario.

Dr. I. M. Halperin announces the opening of an office for the exclusive practice of orthodontics at 843 Queen Anne Road, Teaneck, N. J.

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Secretary-Treasurer, Max E. Ernst _ _ _ 1250 Lowry Medical Arts Bldg., St. Paul, Minn.
Public Relations Bureau Director, Dwight Anderson
_ _ _ _ _ _ _ _ _ _ _ _ _ _ _ _ _ 292 Madison Ave., New York, N. Y.

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Washington-Baltimore Society of Orthodontists

President, George M. Anderson _ _ _ _ _ _ _ _ _ _ 831 Park Ave., Baltimore, Md.
Secretary-Treasurer, Stephen C. Hopkins _ _ _ _ _ 1726 Eye St., Washington, D. C.

American Board of Orthodontics

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Foreign Societies†

British Society for the Study of Orthodontics

President, S. A. Riddett
Secretary, R. Cutler
Treasurer, Harold Chapman

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*The Journal will make changes or additions to the above list when notified by the secretary-treasurer of the various societies. In the event societies desire more complete publication of the names of officers, this will be done upon receipt of the names from the secretary-treasurer.

†The Journal will publish the names of the president and secretary-treasurer of foreign orthodontic societies if the information is sent direct to the editor, 8022 Forsythe, St. Louis, Mo., U. S. A.

DR. OREN A. OLIVER OF NASHVILLE, TENN., PRESIDENT-ELECT OF THE AMERICAN DENTAL ASSOCIATION.